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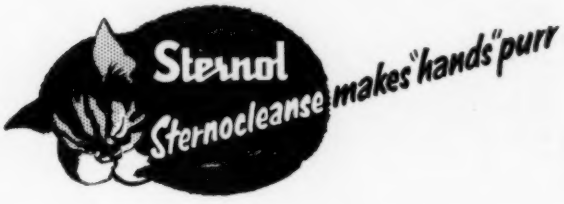

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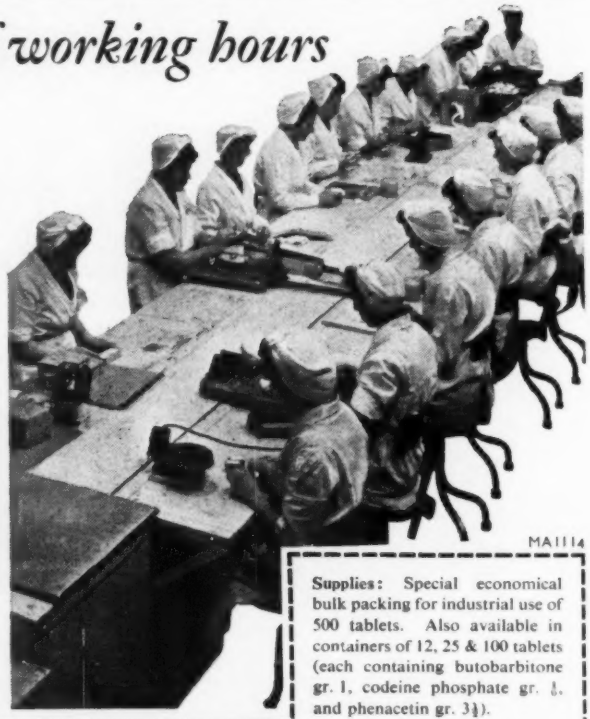

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THE PREVALENCE OF PNEUMOCONIOSIS AND TUBERCULOSIS AMONG EARTHENWARE TOWERS

BY

E. POSNER

From the Stoke-on-Trent Mass Radiography Centre

(RECEIVED FOR PUBLICATION AUGUST 23, 1955)

In the manufacture of general earthenware in Great Britain, towing means the process of smoothing the edges and surfaces of plates, dishes, saucers, etc., in the unfired " green " stage. The articles are placed on rapidly rotating, power-driven wheels provided with hoods and local dust extraction of various designs. The edges are smoothed with tow and pieces of flannel (Fig. 1). In the American ceramic industry and in the tile section of the north Staffordshire pottery industry the process is called " fettling " or " finishing ", and in both countries the work is done almost entirely by women.

The dust arising from the process is that of the unglazed, unfired, brittle earthenware-body, the composition (Rosenthal, 1949) of which is approximately as follows :—

Blue ball clay	18–47%
China clay	24–37%
Flint	21–38%
Cornish stone	7–15%

Ball clays consist mainly of kaolinites (Al_2O_3, $2\,SiO_2$. $2H_2O$), a decomposition product of felspar. They contain some fine grained quartz and iron and lime impurities.

China clays are mineral kaolinites with only few impurities. Flint, as used in English earthenware, is cryptocrystalline free silica containing about 1% of water and a small amount of organic matter. It is broken up by calcining, milled into a fine powder, and mixed with the other parts of the earthenware body. Cornish stone consists mostly of coarsely crystalline felspar and quartz quarried in rock form and finely ground before use (Keeling, 1953).

Although towing has for long been known amongst potters as a very dusty occupation, little information about the prevalence of chest diseases, particularly pneumoconiosis, has so far been published. This lack of statistical information seems to be due to the exclusion of female earthenware workers from the periodical examinations under the Medical Arrangements Schemes of 1931 and 1939 and the similar provisions under the Industrial Injuries Act. In the earthenware industry comprehensive analyses based on periodical examinations exist only for men (Meiklejohn, 1947, 1949a).

In his classical account of dust diseases in the north Staffordshire pottery industry, Arlidge (1892) mentions " the particularly unhealthy conditions " in towers' shops. At that time *x*-ray evidence of pneumoconiosis in the pottery industry was, of course, not available.

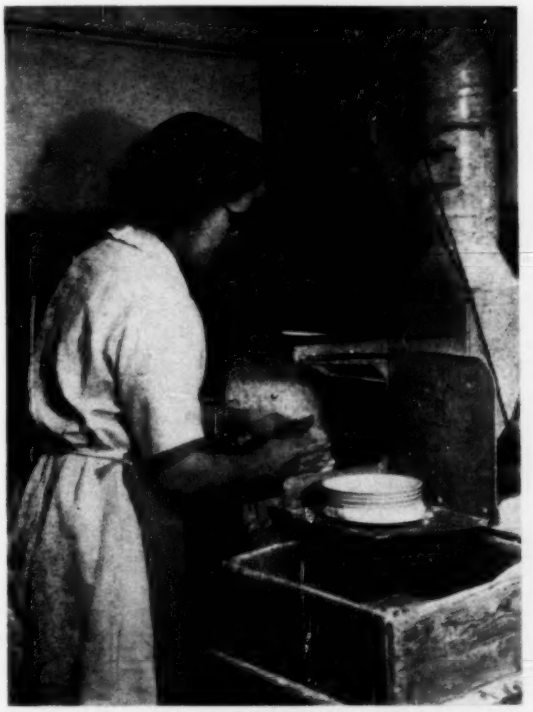

FIG. 1.—Earthenware tower working with an experimental model of a new towing hood designed by the British Ceramic Research Association. *By courtesy of B.C.R.A.*

Sutherland and Bryson (1926) first showed the seriousness of dust disease in the pottery industry by a planned investigation using radiography. The enquiry included 17 towers, one of whom showed radiological signs of silicosis and three evidence of fibrosis, which, it may be assumed, would today be recognized as cases of simple pneumoconiosis of the mixed dust type.

After the introduction of workmen's compensation for scheduled workers in the pottery industry Middleton (1936) analysed the deaths due to silicosis in pottery workers between the years 1930 and 1934. Of nine female operatives, in whom death was considered at necropsy to be due to silicosis, three were towers. This appears to be a high ratio, as only about 10% of all women employed in the clay departments of the earthenware industry are towers.

In the United States, where ceramics, similar in composition to English earthenware, are produced, in Virginia, Flinn, Dreesen, Edwards, Riley, Bloomfield, Sayers, Cadden, and Rothman (1939) found high morbidity rates for silicosis in a group of 83 fettlers. In a larger series Noe (1953) found 25 cases of nodular fibrosis in 214 fettlers and finishers.

Preliminary mention was made in a recent publication (Posner, 1954) of some of the results of examining towers in north Staffordshire. The results are based on mass miniature radiography. As the validity of this method is often considered to be doubtful, the response to these surveys in the pottery industry and the method of the investigations must be described in some detail.

The Response of Volunteers

The 457 earthenware towers in this series attended at 119 factory surveys. Because employment data were not everywhere available, it was not possible to compare this figure with the total number of towers employed at the same time in Stoke-on-Trent, but in 33 factories, chosen at random, the number of towers on the pay roll was 268, of whom 192 attended.

This response of 72·4% is almost the same as that by all women operatives in the north Staffordshire pottery industry, of whom 25,458 out of 34,834 attended mass miniature radiography surveys (73·1%). Elsewhere I have shown (Posner, 1954) that the lapse rates by middle-aged and elderly women in the dust and clay departments of the industry were significantly lower than those in the decorating departments of the same factories. Since pneumoconiosis in occupations in potteries rarely becomes radiologically manifest before the age of 35 years (approximately 20 years of occupational risk), this relatively good response by higher age groups is of great importance. From a comparison with a wage census in 1954, which gives the number of towers over the age of 21 as 445, it is estimated that the response by towers over the age of 35 was in fact nearer 80% than 70%. The sample, therefore, exceeds considerably the number examined by Sutherland and Bryson and by the American authors.

Industrial Histories

The taking of comprehensive industrial histories is frequently neglected at mass miniature radiography routine surveys (Cochrane, 1951). This has been avoided at Stoke-on-Trent surveys, where the emphasis at factory sessions was entirely shifted from clinical to occupational data and all booking-in clerks were well trained in the procedure.

The Use of 35-mm. Film

There has been increasing evidence that the reading of radiographs by a single observer, particularly with regard to pneumoconiosis, is liable to considerable errors and variation of interpretation (Fletcher and Oldham, 1949 ; Cochrane, 1951) and these errors and variations increase with diminishing film size (Fletcher, 1952 ; Newell and McCallum, 1954). It is, therefore, necessary to emphasize that the present results originate from routine mass miniature radiography surveys and not from a research project to investigate the prevalence of pneumoconiosis in towers. At the time of the surveys (1952–54) dual independent reading of miniature films had not yet been introduced into routine mass radiography in this country, but intra-individual dual reading of all rolls of films, which contained radiographs of towers together with a larger number of films of other female clay workers, was arranged.

As a result, 115 towers, or 25·1% of the total, were recalled for full-sized films. The proportion of recalled female plate-makers and casters in the same age groups was very similar. With the exception of seven persons, all recalled towers were over the age of 35.

Despite this high rate of recall, it is highly probable that some cases of early simple pneumoconiosis were missed on the miniature films and that the absolute prevalence rates, shown in Tables 1 and 2 for each group, err by understatement. I would, therefore, suggest that this and similar investigations should be considered no more than a general guide to the relative prevalence in the many and varied occupations, each involving a varying degree of risk from dust in certain industries. Of such industries, pottery manufacture, an industry of many skilled artisans with a large number of

different occupational risks, is an example *par excellence*. As comprehensive examinations using large film of female earthenware operatives have been absent in the past and are not likely to be introduced in the near future, this guide to relative prevalences and risks amongst these women is important.

The Reading of Full-sized Films

In all 60 cases shown in Table I at least two out of three observers agreed in a diagnosis of pneumoconiosis or pneumoconiosis accompanied by tuberculosis. I suspected originally the presence of pneumoconiosis in 73 patients, who were advised to make a claim to the Pneumoconiosis Board. Only 65 did so, of whom 54 (83%) were certified by the Board. The films and industrial histories of those women who failed to submit a claim (eight cases) and of those who were not certified by the Board (11 cases) were sent to Dr. Meiklejohn for his opinion. He expressed a positive opinion on the radiological presence of pneumoconiosis in six of these patients, five of whom had not attended the Pneumoconiosis Board. These six cases, agreed by two observers, were added to the 54 certified cases, giving the total of 60. Three films of poor quality, which could not be repeated, were excluded from further analysis.

The Classification of Full-sized Films

Pathological investigations by Gloyne (1951) in English and by Kirch (1953) in Bavarian potters have shown that the type of mixed dust pneumoconiosis is common in both. The Bavarian porcelain workers handle similar material to English potters (Schneider, 1955, personal communication). It is, therefore, not surprising that many radiographs of English earthenware workers do not present the typical appearances of classical silicosis but a great variety of minute (less than 5 mm.), unevenly distributed, irregularly shaped, fluffy or angular and strandy shadows. An accurate classification of these films according to the scheme of the I.L.O. conference in Sydney (Cochrane, Davies, and Fletcher, 1951) presents considerable difficulties and in this analysis no further subdivision into categories of simple pneumoconiosis has been attempted.

The radiographs of potters with progressive massive fibrosis present the same difficulties of differential diagnosis between that condition and active pulmonary tuberculosis as those of coalminers (Cochrane, Cox, and Jarman, 1952). It is worth while recalling that Gloyne (1951) found pathological evidence of tuberculosis in 43% of potters' lungs, and that Meiklejohn (1949b) has said "tuberculosis may become manifest, even when the degree of silicosis is minimal".

For the present analysis only those cases of tuberculosis were accepted as active which the area chest physicians graded and notified A or B, I, II, III, according to the classification of the Ministry of Health. The criteria for the inclusion in these groups were sputum tests and the usual clinical signs, such as loss of weight and raised sedimentation rates. The same chest physicians assessed the cases during the whole period of the investigation and it is unlikely that their standard of assessment altered during that time.

Prevalence of Pneumoconiosis

The radiological prevalence of pneumoconiosis amongst earthenware towers according to periods of occupational risk is shown in Table I. There are no cases with an employment history of less than 10 years. Between 10 and 19 years the prevalence rate is still low (3·2%) but it rises steeply afterwards to over 30% in women with more than 40 years' exposure.

TABLE I

PREVALENCE OF RADIOLOGICALLY MANIFEST PNEUMOCONIOSIS IN 457 EARTHENWARE TOWERS ACCORDING TO PERIOD OF OCCUPATIONAL RISK

Period of Employment in Years	Less than 10	10–19	20–29	30–39	40 and over	Total
Number *x*-rayed ..	98	93	111	118	37	457
Simple pneumoconiosis	—	2	13	17	10	42
Progressive massive fibrosis	—	1	6	5	2	14
Silicosis accompanied by tuberculosis ..	—	—	—	4	—	4
Total pneumoconiosis..	—	3	19	26	12	60
Morbidity rate (%) ..	—	3·2	17·1	22·0	32·4	13·1

In Table 2 the prevalences of all stages of radiologically manifest pneumoconiosis in towers with more than 20 years' employment are compared with those of other female earthenware workers, who were examined in the same factories and under the same conditions as the towers. The rates for towers are strikingly higher.

TABLE 2

PREVALENCE OF RADIOLOGICALLY MANIFEST PNEUMOCONIOSIS IN SOME GROUPS OF FEMALE EARTHENWARE WORKERS WITH OCCUPATIONAL HISTORY OF MORE THAN 20 YEARS

Group	No. Radiographed	Pneumoconiosis	Morbidity Rate (%)
Plate and saucer makers including assistants	468	50	10·7
Cup handlers and spongers ..	401	18	4·5
Casters and casters' spongers..	602	45	7·4
Towers	266	57	21·4

Comparison with investigations by other authors (Flinn and others, 1939 ; Noe, 1953) are impossible, as they used a different technique on a differently selected working population. It may, however, be significant that Flinn and others (1939), who found higher rates in 1939, commented on the absence of efficient ventilating systems in American fettlers' shops. In England local dust extraction and the provision of hoods over towers' wheels have existed for many years although, as will be shown later, some of the methods of dust extraction have been far from efficient. The amount of dust breathed by English towers is likely to have been less in the past than that by American fettlers before 1939.

Active and Infectious Pulmonary Tuberculosis

As the result of the mass radiography surveys, seven towers were notified as suffering from active pulmonary tuberculosis. Three cases (ages 21, 25, and 48) showed no definite radiological evidence of pneumoconiosis and in four cases (ages 51, 53, 55, and 58) there was a definite pneumoconiotic background. In each group there were two sputum-positive cases (21 and 48 years and 48 and 53 years old respectively).

Table 3 shows the rates of active pulmonary tuberculosis in towers compared with those of other earthenware clay workers, biscuit-warehouse workers, dippers, decorators, and finally with women in Stoke-on-Trent who had never been employed in any job in the pottery industry. The criteria for the assessment of activity (see above) were the same for all groups. Although the number of towers and other clay workers, shown in Table 3, cannot be accurately compared by strict statistical standards, the double rate for towers, compared with other clay workers, is at least strongly suggestive of a true higher prevalence. The differences between towers and earthenware clay workers on the one hand and pottery decorators and women not connected with the pottery industry on the other seem to be statistically highly significant. Pottery decorators share with clay workers the general occupational hazards of factory life, particularly those of crowded workshops, but they are not exposed to a specific risk from dust.

TABLE 3

PREVALENCE OF ACTIVE PULMONARY TUBERCULOSIS IN VARIOUS GROUPS OF WOMEN EARTHENWARE WORKERS AND WOMEN UNCONNECTED WITH POTTERY INDUSTRY OF ALL AGES IN STOKE-ON-TRENT 1952–54

Category	No. Radiographed	Cases of Active Tuberculosis	Rate per 1,000
Towers	457	7 (4)*	15·3
Clay workers (except towers) ..	3,892	29 (9)	7·4
Biscuit-warehouse workers ..	940	5 (2)	5·3
Dippers	686	3 (—)	4·1
Decorators and glost warehouse	9,271	24 (8)	2·6
Stoke-on-Trent residents not connected with pottery industry	10,045	26 (9)	2·6

* The figures in brackets are cases with positive sputum. They are included in the total and are not additional.

Dust Conditions and Prevention

The Pottery Health and Welfare Regulations (Ministry of Labour, 1950) consolidated and enlarged the various provisions which had been made in the past by statutory orders (Special Regulations, 1931, 1932, and Factories Act, 1947). Towing is one of the processes scheduled in the 1950 Regulations " which should not be carried out without efficient exhaust draught ". In 1951 the British Ceramic Research Association began to investigate dust conditions in towers' shops and at the benches and to test the efficiency of existing designs of hoods and local exhaust ventilation. One of the investigators was W. A. Bloor, who, together with others, had studied dust concentrations in earthenware clay shops before the war but not specifically those related to towers (Bloor, Goodall, and Webb, 1938). The distribution and movement of dust were studied by cinematography with special lighting, which revealed dust clouds invisible under normal conditions. This is a method not only of great technical interest, but also of great demonstrative and educational value to everybody concerned with dust suppression in factories. Quantitative data were obtained by using a thermal precipitator. A number of towing hoods of traditional design were tested, and a new design of hood, developed by the Research Association, was installed in a number of factories and similarly tested. The quantitative data resulting from these investigations are shown in a condensed form in Table 4. All particles shown in Table 4 were of the size 0·5 to 5 microns (Bloor, 1955, personal communication). The reduction of concentration of dust by the new designs and even more of spillage of dust on to the floor is impressive.

TABLE 4

DUST CONCENTRATIONS IN TOWERS' SHOPS

Hood	Particles per c.cm. (0·5–5 micron)		Spillage of Dust on Floor (g./hr.)
	At Breathing Level of Worker	In Surrounding Atmosphere	
Various old types of towers' hoods.. ..	144–310	72–250	15
New hood designed by British Ceramic Research Association ..	28–43	22–40	Less than ½

The principles of the new hood (Fig. 1), which has now been recognized by H.M. Factory Inspec-

tors as complying with the existing regulations, are briefly these.

(1) It provides a streamlined airflow into and through the hood.

(2) The towing wheel is so positioned with respect both to the exhaust outlet and the front that the exhaust air is used with maximum efficiency and spilling from the hood is avoided.

(3) The specified minimum horizontal airflow towards the outlet, measured at 2 in. above the centre of the ware, without the air-jet in operation, is 300 ft. per min.

(4) A pressure controlled air-jet directed on to the ware safely removes the small amount of loose dust that remains on the surface. This eliminates the necessity for the undesirable practice of " mouth blowing " by the tower.

(5) The side platforms for the accommodation of supplies of ware are so designed as to induce an appreciable airflow over the ware, ensuring that any fine dust produced in this region will be taken into the hood and not dispersed into the general atmosphere of the workroom.

A considerable number of women will remain employed as towers in the north Staffordshire industry and the design of more efficient dust suppression at their working places seems to be an important step forward. At the same time it must not be forgotten that many other measures, such as improved layout of workshops, mechanization, obligatory provision of protective garments, vacuum cleaning of workshops, etc., which have now been widely introduced, should play a most important part in the reduction of pneumoconiosis amongst earthenware towers. The benefits of these improvements can only be assessed after a considerable number of years by periodical *x*-ray examinations of towers, particularly of those who started to work under the much improved hygienic conditions after the last war.

Summary

Routine mass radiography surveys in Stoke-on-Trent revealed high prevalence rates of radiologically manifest pneumoconiosis and of active tuberculosis amongst female earthenware towers.

The limitations of 35 mm. *x*-ray examinations with regard to true prevalence rates and their value with regard to relative prevalence rates in certain industries are discussed.

The principles of newly introduced protective appliances at towers' benches are shown.

I am very grateful to Dr. A. T. Green, O.B.E., Director of the British Ceramic Research Association, for permitting me to use material collected by himself and his staff, and to Messrs. A. Dimsdale and W. A. Bloor who kindly criticized and corrected the technical part of this paper.

Dr. A. Meiklejohn once again let me have the benefit of his great experience with pneumoconiosis amongst potters. Dr. F. A. Smith and Dr. E. Bennion, consultant chest physicians, kindly allowed me free access to their records. I received much helpful advice from Mr. E. Waller, H.M. Factory Inspector at Stoke. The Secretaries of the British Pottery Manufacturers' Federation and of the Hanley Employment Exchange provided useful employment data. Dr. K. W. Cross, Senior Statistical Officer of the Birmingham Regional Hospital Board, very kindly gave his opinion on the significance of tuberculosis rates.

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A REPORT ON THE ABSENCE OF PNEUMOCONIOSIS AMONG WORKERS IN PURE LIMESTONE

BY

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It is generally believed that exposure to limestone dust does not present a pneumoconiosis hazard. However, Doig (1955) has recently described cases of silicosis amongst limestone workers after prolonged exposure to high dust concentrations. The limestones concerned contained from 5 to 15% quartz as impurity, and dissolution of the calcium carbonate in the lung would leave a quartz-rich residue which may well have caused the disease. If this interpretation were correct no pneumoconiosis should occur amongst workers in pure limestone. It was decided to test this by radiological and clinical examination of men who should have been exposed to high dust concentrations of pure limestone for long periods of time.

The purest limestone found in sufficient quantity for commercial use in the British Isles occurs in the Buxton area of Derbyshire. This limestone is of the Lower Carboniferous age and contains upwards of 97% of calcium carbonate. Its quartz content is usually below 1%. There are many quarries and grinding plants in the area but the basic processes are identical in most plants. The quarried rock is broken by crushers, screened, and dried. The dried product is pulverized and bagged. Dust conditions vary with the type of machinery and ventilation systems in use. In general, the smaller and older plants are more dusty than the large modern ones. Rotary driers, pulverizers, and bagging machinery produce most of the dust but crushers and conveyors also can be very dusty. The ventilation systems usually discharge the dust into the open air above roof height, and its further dispersal is very much affected by wind and weather. There is a definite haze of dust in many plants and a covering of dust over machinery, ladders, and floors. The faces, hands, and clothing of the workers are covered by a layer of dust.

In the course of the survey made in the autumn of 1954 a large number of quarries and grinding plants were visited.

The names of 29 men with histories of sufficiently long exposures to pure limestone dust were obtained. They were interviewed individually and occupational histories were taken. Fifteen had to be rejected because they had worked either with gritstone, chert, igneous rock, or in a brickworks at some time or other. This left a group of 14 men for clinical and *x*-ray examination; they had been exposed to heavy dust concentrations for periods varying from 12 to 35 years and had worked at eight plants given in code letters A to H in Tables 1, 2, and 3.

The 14 men were examined at the Manchester Royal Infirmary. Full-size radiographs were taken, and thorough clinical examinations were made. The main result of the work was that no trace of abnormalities due to dust could be seen on the radiographs nor was there any clinical sign of pneumoconiosis or of chronic bronchitis. Detailed results are given in Table 1.

Dust Composition and Concentration

Samples of fine dust were obtained from the eight plants and analysed for quartz content; the results are given in Table 2, from which it will be

TABLE 2

QUARTZ ANALYSIS OF LIMESTONE SAMPLES

Source	Acid Insoluble (%)	Quartz in Sample (%)
Plant A	0·3	0·1
,, B	3·9	1·1
,, C	2·3	0·3
,, D	1·4	0·3
,, E	1·7	0·2
,, F	0·8	0·2
,, G	0·5	0·2
,, H	0·4	0·2

TABLE 1

CLINICAL FINDINGS OF 14 MEN EXPOSED TO PURE LIMESTONE DUST

Plant	Worker	Age	Exposure (years)	Occupational History	Past Medical History	Clinical Examination	Radiograph of Chest
A	J.P.	51	35	Has been in every process of industry and heavily exposed to limestone dust	In the past had 2 years gastric illness but apart from this nothing else	Chest N.A.D. Chest expansion 2 in. B.P. 130/80 mm.Hg. Weight 11 st. 8 lb.	Normal
A	T.A.G.	51	35	Has always been a quarryman: fitter's mate, crusherman, dust tipping (quarry spoil), quarry face (stone filler), platelayer (for tubs), plant attendant	Nil	Chest N.A.D. Chest expansion 3 in. B.P. 130/80 mm.Hg. Weight 12 st. 1 lb.	Normal
A	S.I.W.	58	33	Maintenance fitter in plant. Heavy exposure to dust	Pneumonia at age 30, otherwise no serious illnesses	Chest N.A.D. Chest expansion 3 in. B.P. 180/100 mm.Hg. Weight 12 st. 4 lb.	Normal
A	J.S.W.	52	27	Plant attendant	Nil	Chest N.A.D. Chest expansion 2 in. B.P. 140/80 mm.Hg. Weight 12 st. 5½ lb.	Old scarring in L. apex. No active lesion
B	R.H.F.	39	12	General foreman: has done all jobs in the plant	Some 12 months ago began to get symptoms (of a psychoneurotic type) of being unable to take a deep breath, etc. Was radiographed at various places, nothing found. Reports that he has improved considerably over the last few months	Chest N.A.D. Chest expansion 4½ in. B.P. 120/75 mm.Hg. Weight 11 st. 3½ lb.	Normal
C	F.H.B.	76	23	Until the age of 52 worked in the engineering trade. Worked as a plant attendant until a year ago (when he was 75 years of age). Heavily exposed to dust	Nil	Remarkably youthful man for his age. Chest, occasional rhonchi heard. Chest expansion 3 in. B.P. 200/90 mm.Hg. Weight 12 st. 5 lb.	Both lung fields show a rather exaggerated pattern throughout and the lung markings extend peripherally farther than normal. Within the normal limits for his age
D	T.A.	52	35	Trimming, lettering, and machining limestone	At 17 years of age had pleurisy with effusion. Off 10 months. No further illness	A youthful 52. Chest N.A.D. Chest expansion 3 in. B.P. 190/110 mm. Hg. Weight 12 st. 10 lb.	There is an adhesion in the right costophrenic angle; the lung pattern is otherwise normal
E	R.L.A.	41	27	Plant attendant and bagger	At 14 years of age had bronchitis with measles	Chest N.A.D. Chest expansion 2 in. B.P. 120/80 mm. Hg. Weight 9 st. 12 lb.	Normal
E	J.F.	46	25	Plant attendant and bagger	Nil	Chest N.A.D. Chest expansion 2½ in. B.P.145/110 mm.Hg. Weight 10 st. 9 lb.	Normal
F	W.G.	35	16	Limestone bagger	Nil	Chest N.A.D. Chest expansion 3 in. B.P. 130/90 mm.Hg. Weight 11 st.	Normal
G	W.H.P.	55	34	Limestone crusherman	Nil	Some kyphosis. Chest N.A.D. Chest expansion 2 in. B.P. 210/130 mm. Hg. Weight 10 st. 8 lb.	Left diaphragm higher than the right. No pulmonary lesion
G	G.W.E.	52	27	Plant attendant, bagger, and crusher	Nil	Chest N.A.D. Chest expansion 2½ in. B.P. 140/85 mm.Hg. Weight 10 st. 10 lb.	Normal
H	C.C.G.	44	21	Plant attendant	Nil	Chest N.A.D. Chest expansion 1½ in. B.P. 130/85 mm.Hg. Weight 11 st. 6 lb.	Normal
H	J.W.H.	47	20	Plant attendant	Nil	Marked kyphosis. Chest N.A.D. Chest expansion 3 in. B.P. 130/75 mm.Hg. Weight 8 st. 12 lb.	The left costophrenic angle blunt. No pulmonary lesion

seen that one sample had 1·1% of quartz and all others had less than 0·5%.

At three of the plants thermal precipitator samples were taken during working hours. These samples were taken at breathing level at places where men worked or passed, usually near a source of airborne dust. At two of the plants two thermal precipitators were used, sampling at approximately the same time within a few feet of each other.

The thermal precipitator samples were evaluated to give the concentration of particles within the size range 0·5 to 5 microns (Table 3). With some of the samples a complete size distribution of the particles was measured.

TABLE 3

THERMAL PRECIPITATOR DUST COUNTS AT THREE LIMESTONE PLANTS

Plant	Sampling Point	0·5–5 μ particles per cc.	
		(I)*	(II)*
A	Mixing platform	1,740	355
	Discharge from secondary crusher	560	555
	Above coarse dust bunker	2,350	3,350
	Vibratory screen platform	1,250	2,030
	" " "	—	1,470
	Two-foot cone crusher	1,560	1,790
	Discharge from vertical driers	690	790
	Loading and stacking bags	240	310
	Between mills	3,640	2,420
D	Below primary crusher hopper	590	460
	Above rotary screen	515	60
	Discharge from secondary crusher above rotary drier	3,620	2,450
	Discharge to screens from rotary drier above jaw crusher	3,240	2,280
	At jaw crusher	5,920	5,390
	Bagging from cyclone	760	470
F	Top screens	4,480	—
	Top Mill—middle floor pulmac mills	5,390	—
	Top Mill—first floor	5,030	—
	Top Mill—ground floor near bagging operator	3,480	—
	Top Mill—basement discharge from rotary drier	3,620	—
	Bottom Mill—near bagging operator	2,650	—

* I and II are duplicate samples.

Many of the samples showed high concentrations of dust, and those at plant F were of the order of 3,000 to 5,000 particles per cc. Even at Plant A, where extensive dust extraction equipment was installed, some of the dust counts were quite high. These results indicate the level of airborne dust concentrations to which workmen in the plants are exposed. The concentrations may vary appreciably with time, depending mainly on the amount and direction of ventilation and the degree of activity.

The size distributions were typical of airborne dust. In all cases over 80% of the particles were below 5 microns in size, and over 20% below 0·25 micron.

Comment

Dust concentrations during the last 30 years are not known but it is very likely that they were higher than those found at the present time when much attention is paid to dust suppression. Nevertheless, many of the figures measured now showed dust counts of the order of 3,000 particles per cc. of a size between 0·5 and 5 microns. The quartz percentage of this dust was, however, extremely low. There was no sign of accumulated dust in the lungs of any of the men examined and no clinical evidence of bronchitis. It appears very likely that calcium carbonate can be eliminated from the lungs, probably by dissolution as calcium bicarbonate. It follows from these observations that long exposure to extremely high dust concentrations of pure limestone does not lead to any fibrosis of the lung.

Summary

A clinical and an *x*-ray examination were carried out on 14 men who had worked in pure limestone for periods varying from 12 to 35 years ; no evidence of pneumoconiosis was found although the dust counts showed many thousands of particles between 0·5 and 5 microns in the atmosphere to which the men were exposed.

This survey would have not been possible without the wholehearted cooperation of management, union officials, and workers at the quarries.

We wish to thank Professor R. E. Lane, Nuffield Department of Occupational Health, Manchester University, and Dr. E. D. Gray, radiologist, Manchester Royal Infirmary, for the clinical and radiological investigations ; our thanks are also due to Mr. R. H. Griffiths, H.M. Inspector of Quarries, for help in selecting the quarries ; to Mr. W. Staton, H.M. Senior Inspector for Special Development Duties (Dust) ; to Dr. J. T. Burdekin and Mr. S. R. Asquith, Safety in Mines Research Establishment, for doing the dust counts, and to the Ministry of Fuel and Power for permission to publish. The cost of this investigation was met by a grant from the Medical Research Council.

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FURTHER STUDIES OF THE DUST IN LUNGS OF COAL-MINERS

BY

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In a previous communication (King and Nagelschmidt, 1945), analytical data were given for the amount and composition of the dust found in the lungs of 54 coalworkers from South Wales. Coal, total silica, quartz, mica, and kaolin were determined as the percentages of dried lung tissue, and the results were related to type of work and degree of pathology. The main findings were the following :

(1) The kind of occupation was clearly revealed in the composition of the dust from the lungs. For instance, its coal percentage varied from 25 for rock-workers* to 95 for trimmers and screen-workers.

(2) The average dust composition in the lungs of 27 anthracite workers was very similar to the composition of the airborne dusts at the coal face, and thus there was no evidence of any mineral change in the lung, as far as the ratio of coal to non-coal or the relative proportions of mica, quartz, and kaolin were concerned.

(3) In the pathological classification three grades were distinguished : reticulation, mixed nodulation, and confluent fibrosis. In addition, three rock-workers were classed as a silicotic nodulation group. The concentrations of coal, quartz, and total silica varied considerably within each pathological group, but increased only very slightly from one group to the next, except for the silicotic nodulation group ; in the more homogeneous group of the 27 anthracite colliers the increases in lung dust components with increasing grade of pathology were not significant at the 95% significance level.

In the discussion of the results it was stated that the silicotic nodulation was due to quartz, but that it could not be decided on the basis of the data whether reticulation was also a quartz effect, modified by the large amounts of coal and other siliceous minerals, or whether it was a type of lung change due to the presence of large amounts of inert foreign material which could have been produced by equivalent amounts of pure coal without the associated siliceous components found in the airborne dusts. Information from lung residue data may not be able to solve this problem, but the dust in the lungs and the associated pathological changes are material evidence which cannot be neglected.

In order to gain further information more lungs have been analysed. In particular, attempts have been made to secure material from the earlier stages of the disease and from the whole range of coal rank found in South Wales. These attempts have only been partly successful, as will be shown later. The present report deals with the analyses of 71 further lungs of coalworkers from South Wales, and of 15 silicotic lungs of tin-miners and granite-workers from Cornwall, which are included for comparison. The 86 new lungs will in the following be called the " new series " in contrast to the 54 lungs described previously, which will be referred to as the " old series ". Data on lungs of three men without known dust exposure have also been obtained.

It was originally intended to pool the information from the coalworkers of the old and new series in the hope that the indicative but not statistically significant results from the old series would become significant for the whole group. Such pooling could, however, not be done, as will be described later, because whole lungs were analysed in the new series as against selected fibrosed parts of lungs in the old one. The present results are therefore again in the nature of a progress report.

Ideally, one would want to measure dust exposures for groups of men and radiograph them periodically. After death, one would examine and analyse the lungs and could then relate radiographs,

* The reference to rock-workers throughout this paper means rock-workers in coal-mines.

pathology, and analyses of dust in the lungs to the dust exposure. This is at present impossible, and the best that can be done is to relate the pathological data to the dust in the lungs and to such industrial histories and radiographs as can be obtained. As the taking of dust records improves and proceeds in British coal-mines, it will be possible, in the future, to have better exposure records for lungs that come to necropsy and analysis. The purpose of the present work is thus to see what relation, if any, there is between the amount of total dust or of any of its components and the type of pathological change found in the lungs.

Source of Material

Professor J. Gough has provided the following note on the source of the lungs and the criteria used in selecting these lungs :

All the necropsies were carried out at the Pathology Department of the Welsh National School of Medicine so that the lungs were immediately available for preparation. This was an advantage as the lungs could be dried without the addition of any fixative or other fluid. The placing of lungs in fixative has a disadvantage in that a considerable amount of dust may be washed out. The necropsies were on men who were known to have had pneumoconiosis, on those in whom it was alleged or suspected that pneumoconiosis was present although this had not been diagnosed during life, and also on miners dying in general hospitals from causes other than pneumoconiosis. The aim was to get an equal number of lungs from various grades of pneumoconiosis. A sufficient number of cases of massive fibrosis were quickly obtained, but the cases of simple pneumoconiosis were obtained over a much longer period. (Further details on the selection of lungs are given in the discussion.) The right lung was used for chemical analysis except for small portions reserved for histology and bacteriology. The hilar glands and main bronchi were excised and discarded and the lung tissue was then cut and placed in pyrex jars, dried at approximately 105° C., and weighed. The left lung was used for preparing large sections.

There is one important difference in technique between the old and the new series. In the old series representative parts of lung tissue, usually amounting to about 200 g. wet weight, were selected, dried, and analysed. It was therefore only possible to give dust concentrations (per cent. dry lung), but not the absolute amounts per lung. In the new series the whole of both lungs was available. The left lung in every case was used for pathological examination and for cutting large sections by the technique of Gough and Wentworth (1949). A representative sample of the whole right lung was used for analysis, and concentrations and absolute amounts of the total dust and of the different dust constituents were determined.

Badham and Taylor (1938) have discussed the problem of lung sampling fully ; they cut the lungs in an axillary direction and used one half of the left and one half of the right lobes for analysis. But the cutting of the lungs either fixed or unfixed inevitably leads to the loss of some dust. In our opinion the analysis of the right (or left) lung is preferable, unless there are gross disparities between the two lungs. Such lungs were not included in the series.

Classification

In order to retain conformity with the old series the same pathological classification was used as before, but one earlier grade, slight reticulation, was added.

We therefore have the following five pathological grades : (1) slight reticulation, (2) reticulation, (3) mixed nodulation, (4) confluent fibrosis, and (5) silicotic nodulation.

Pictures of representative large sections from lungs in each of the categories are reproduced in Figs. 1 to 5. (We are indebted to Professor J. Gough for these illustrations.)

The radiological classification used was that of the Pneumoconiosis Research Unit described by Cochrane, Davies, and Fletcher (1951), which distinguishes three grades of simple pneumoconiosis and four grades of progressive massive fibrosis (P.M.F.). Its relation to the pathological grades is described on p. 16.

The occupational groups are also the same as in the old series, except that no separate groups of anthracite- and steam-coal colliers could be made, as most of the colliers had worked in steam-coal mines. A further group, " unclassifiable underground ", had to be added which reflects the failure to obtain adequate working histories. All the groups are from South Wales except the last group of tin- and granite-workers from Cornwall, where

TABLE I
NUMBER OF LUNGS IN NEW SERIES ACCORDING TO OCCUPATION AND PATHOLOGICAL GRADING

Occupation	Pathological Grade					Total
	1	2	3	4	5	
Rock-workers	1	1	3	4	—	9
Firemen-shotsmen	—	—	—	3	—	3
Hauliers, riders, etc.	4	3	2	—	1	10
Colliers	1	8	9	10	—	28
Workers on surface screens	2	1	—	—	—	3
Not classifiable	3	7	6	2	—	18
Tin-miners and granite-workers	1	—	—	—	14	15
Total	12	20	20	19	15	86

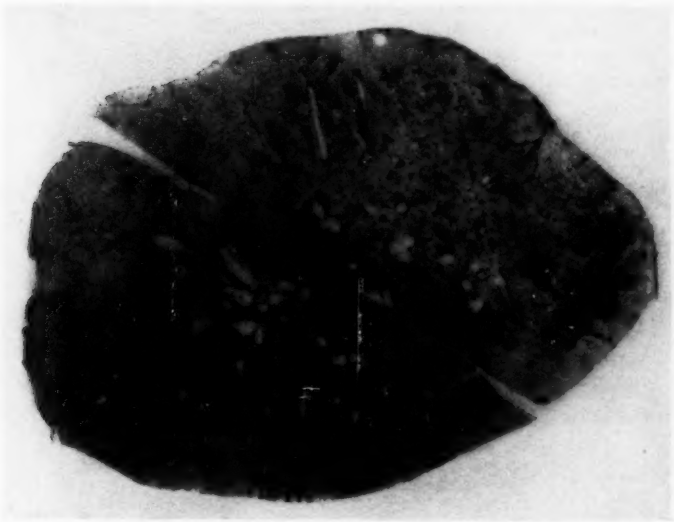

FIG. 1

FIG. 1.—Slight reticulation.
FIG. 2.—Reticulation.
FIG. 3.—Mixed nodulation.

FIG. 2

FIG. 3

FIG. 4.—Confluent fibrosis.
FIG. 5.—Silicotic nodulation.

FIG. 4.

FIG. 5.

the type of disease produced is usually classical nodular silicosis ; this series was analysed in order to provide a contrast to the pneumoconiosis of coal-miners.

The distribution of the new series in terms of working histories and degrees of pathology is shown in Table 1.

Coal varies in rank, and for South Wales' coals this variation can be adequately described by changes in the volatile matter content.

In conformity with the previous work we have ranked the coal as follows :—

Symbol	Name	Volatile Matter (%)
A	Anthracite	4–10
S	Steam coal	10–22
B	Bituminous coal	22–36

Some workers had mixed histories in so far as they worked for several years in different types of coal.

Table 2 shows for the whole group from South Wales and for the more homogeneous group of colliers how many worked in coals of different rank.

It will be seen that most of the South Wales lungs came from persons who had worked in steam-coal mines. In the group of colliers there were 20 from the steam-coal areas, three from bituminous, none from anthracite mining. The remaining five had had mixed exposures. This is unfortunate as more information, especially for bituminous-coal workers, is required. In spite of all efforts, however, it has up to the present not been possible to obtain sufficient lungs from bituminous and anthracite areas.

TABLE 2

NUMBER OF COALWORKERS' LUNGS IN NEW SERIES ACCORDING TO RANK OF COAL WORKED AND PATHOLOGICAL GRADING

Rank of Coal	All Cases from South Wales						Colliers Only				
	Pathological Grading					Total	Pathological Grading				Total
	1	2	3	4	5		1	2	3	4	
A	—	1	—	—	—	1	—	—	—	—	—
A, S	1	1	1	1	—	4	—	—	1	1	2
S	7	9	13	15	1	45	1	5	7	7	20
S, B	—	1	1	1	—	3	—	—	—	1	1
B	—	3	2	—	—	5	—	2	1	—	3
A, S, B	—	—	—	1	—	1	—	—	—	1	1
Unknown ..	3	5	3	1	—	12	—	1	—	—	1
Total ..	11	20	20	19	1	71	1	8	9	10	28

A = Anthracite, B = Bituminous coal, S = Steam coal.

Methods of Analysis

On removal from the body the whole right lung was cut into pieces and dried in an air oven at approximately 105° C. in Professor Gough's laboratory at Cardiff. The dried lung was then sent to the Postgraduate Medical School of London. Here it was broken into small pieces, again dried at 105° C., ground to a fine powder in a power-driven mortar, sieved through 60 B.S.S. screens, re-ground, and further dried to constant weight. It was then well mixed and sampled by " quartering " for determination of " coal " by the method of King and Gilchrist (1945) and ashed for chemical and *x*-ray analysis. For ashing, 50 g. of the dried powder was placed in shallow " vitreosil " dishes (4 in. by 6 in.) and heated in a thermostat-controlled muffle furnace at 380° C. until no black material remained. This required usually five to six days. The light fluffy ash which was free from carbon particles was now carefully brushed on to a 15 cm. filter paper, Whatman No. 541, by means of a camel hair brush. The filter paper was folded over the ash, placed inside a large Gooch filter and held in place by a wad of cotton wool. By means of a continuous liquid extractor the ash was washed twice with cold N HCl for 24 hours and then washed with water. This was found to be adequate to remove completely all the Fe''', PO_4''' and CO_3'' of the indigenous lung ash from the mineral dust and was not appreciably destructive of the siliceous minerals, as shown by the fact that only small amounts of silica were found in the HCl used for extraction (colorimetric method for silica, King, 1939).

The HCl-insoluble residue of the ash consisted of the quartz and other siliceous minerals of the lung. The folded filter paper was removed from the Gooch filter, placed in a platinum crucible, dried, ignited at 380° C. in the same muffle furnace as before, cooled, and weighed. This weight represented the " siliceous minerals " contained in the sample of dried lung. " Coal " plus " siliceous minerals " represents " total dust ".

For silica analysis about 100 mg. of the siliceous minerals was fused with sodium carbonate, and the silica determined gravimetrically after double evaporation with hydrochloric acid by the classical method. The results were calculated to give the percentage SiO_2 in the siliceous minerals and in the dried lung. Alumina was determined in the combined filtrate from the silica determination by the method of Gentry and Sherrington (1946).

The mineral content of the ashed acid-extracted lung residues was determined at the National Physical Laboratory and later also at the Safety in Mines Research Establishment by the *x*-ray diffraction technique using powder diagrams. In contrast to the previous results there was much less, and usually no kaolin in the residues. This was only discovered towards the end of the work. Check repeats with careful temperature control showed that several previously kaolin-free residues did contain kaolin when new samples were ashed at 380° C., and it is likely that the bulk of the samples were ashed at too high a temperature. It was therefore not possible to make estimates of kaolin and mica separately but a joint " kaolin plus mica " estimate could be obtained in all cases. By visually estimating and comparing *x*-ray line intensities, the approximate relative proportions of quartz and kaolin plus mica were found and calculated back to percentages of dry lung on the basis of the total silica values.

Results

The results of the analyses are given in detail in Table 3. The lungs are grouped according to occupation and pathological grade as defined above, and, within each grade, according to increasing amounts of total dust as per cent. dry lung. Age, numbers of years worked in different occupations, and numbers of years elapsed between removal from dust and death are also given.

From the dry weights of the right lung given in the last column of Table 3 the actual amounts of dust in both lungs can be estimated by multiplying the percentages of the different constituents by twice the lung weights.

Effect of Occupation.—The composition of the dust varied with the type of occupation as shown in (A) Table 4 and Fig. 7; the occupations are arranged in increasing order of coal percentages of the lung dusts. The small groups of firemen, screen-workers, and non-miners are omitted from this tabulation, as there are only three lungs in each of the groups. " Coal " in tin-miners' and granite-workers' lungs is the ignitable portion of the residue prepared by alkali digestion. It is probably mainly soot from acetylene lamps, candles, and domestic fires, and

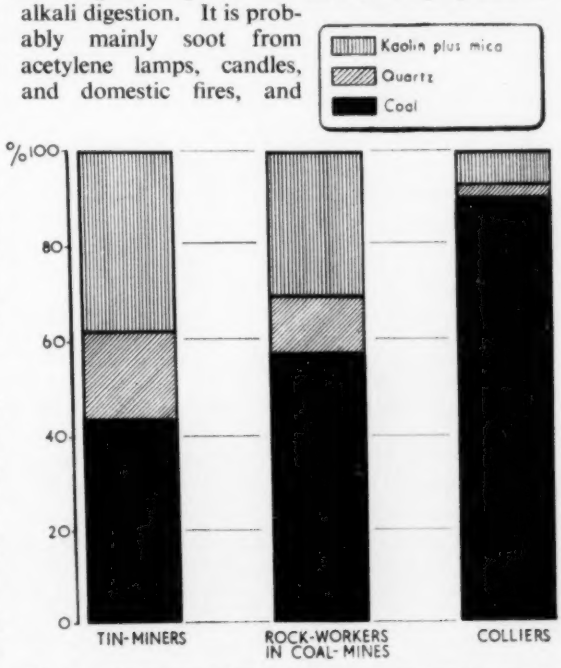

FIG. 7.—Relative proportions of coal, quartz, and kaolin plus mica in the average lung dust of different occupational groups.

TABLE 3

MINERAL CONTENT OF LUNGS OF COALWORKERS AND TIN-MINERS

Chief Occupation	P.M. No.	Age	Duration of Exposure (years)*			Pathological Grade†	Percentage Dry Lung						Dry Weight of Lung (g.)
			1	2	3		Total Dust	Coal	Siliceous Minerals	Silica	Quartz	Kaolin plus Mica	
I. Rock-workers in coal-mines	51	72	20	20 (IV)	2	1	3·46	2·20	1·26	0·84	0·41	0·86	108
	2	58	30	—	2	2	5·40	2·54	2·86	1·80	0·92	1·93	133
	35	67	50	—	2	3	6·21	3·20	3·01	2·03	1·01	2·01	81
	8	67	29	25 (III)	0	3	6·36	4·90	1·46	0·91	0·42	1·03	109
	75	62	26	—	?	3	9·27	5·90	3·37	2·15	0·98	2·38	138
	93	66	22	15 (II)	1	4	3·18	1·27	1·91	1·20	0·48	1·44	223
	94	45	18	7 (IV)	5	4	3·40	1·75	1·65	0·99	0·41	1·22	245
	72	62	14	16 (III)	5	4	8·34	5·30	3·04	1·87	0·75	2·27	143
	77	62	35	3 (IV)	4	4	13·58	6·50	7·08	4·40	1·79	5·28	205
II. Firemen	97	71	55+	—	0	4	5·15	4·10	1·05	0·70	0·34	0·72	136
	9	65	27	23 (IV)	2	4	14·12	12·2	1·92	1·16	0·45	1·44	189
	59	54	18	10 (IV) 4 (III)	5	4	31·41	29·50	1·91	1·22	0·50	1·43	215
III. Miscellaneous underground workers (hauliers, repairers, roadmen)	34	51	30	—	?	1	1·46	1·17	0·29	0·18	0·10	0·17	128
	92	65	27	5 (IV)	0	1	1·79	1·40	0·39	0·23	0·10	0·28	101
	89	69	16	—	10	1	3·70	3·50	0·20	0·13	0·06	0·14	130
	62	59	26	6 (I)	?	1	3·72	3·20	0·52	0·29	0·11	0·39	80
	63	60	34	6 (IV)	5	2	1·10	0·90	0·20	0·13	0·06	0·14	63
	91	61	40	6 (IV)	2	2	3·48	3·10	0·38	0·27	0·12	0·27	90
	27	65	44	6 (IV)	3	2	4·81	3·20	1·61	1·04	0·41	1·21	114
	5	65	52	2	0	3	6·85	5·40	1·45	0·98	0·50	0·95	138
	44	72	42	16 (IV)	0	3	8·62	5·90	2·72	1·75	0·83	1·89	137
	3	45	10	5 (I) 6 (IV)	5	5	5·85	1·37	4·48	2·75	1·92	2·55	183
IV. Colliers	74	40	12	4 (I)	4	1	2·67	2·5	0·17	0·11	0·04	0·13	98
	82	49	12	—	?	2	2·33	2·0	0·33	0·23	0·15	0·18	137
	66	47	27	—	?	2	3·64	3·5	0·14	0·09	0·04	0·11	136
	17	42	14	14 (I)	0	2	4·13	3·5	0·63	0·42	0·21	0·42	156
	79	47	28	—	7	2	5·08	4·6	0·48	0·31	0·13	0·34	189
	25 26	60	30	—	13	2	5·22	4·8	0·45	0·29	0·14	0·31	222
	47	60	40	—	2	2	5·74	4·4	1·34	0·81	0·33	1·01	130
	53	50	36	—	0	2	6·57	6·0	0·57	0·36	0·17	0·41	97
	86	58	38	8 (III)	2	2	12·54	10·2	2·34	1·42	0·58	1·75	98
	7	53	31	—	9	3	7·19	6·4	0·79	0·49	0·21	0·57	200
	61	64	50	—	0	3	7·67	5·5	2·17	1·32	0·52	1·65	131
	57	45	23	—	5	3	8·23	7·3	0·93	0·58	0·26	0·67	140
	4	53	37	—	4	3	8·31	6·4	1·87	1·08	0·41	1·45	155
	6	55	25	11 (III)	7	3	11·47	10·6	0·87	0·54	0·23	0·65	200
	65	44	27	4 (V)	0	3	13·85	12·5	1·35	0·89	0·40	0·96	158
	108	56	27	—	16	3	16·0	14·6	1·40	0·81	0·31	1·09	167
	95	54	32	—	7	3	17·44	16·1	1·34	0·78	0·29	1·04	187
	45	53	24	3 (I) 3 (VI)	10	3	21·07	20·1	0·97	0·64	0·28	0·69	87
	107	54	24	7 (VI)	10	4	4·36	3·5	0·86	0·54	0·24	0·62	128
	96	64	40	3 (VI)	?	4	6·46	5·5	0·96	0·59	0·26	0·70	151
	99	63	42	—	5	4	9·15	8·9	0·25	0·16	0·06	0·19	121
	22	57	36	—	7	4	9·33	7·2	2·13	1·26	0·47	1·65	140
	1	64	19	9 (VI)	1	4	9·99	6·9	3·10	1·91	0·69	2·41	103
	24	60	30	6 (V)	?	4	19·25	18·4	0·85	0·53	0·25	0·59	106
	10	59	29	11 (I)	5	4	19·76	18·3	1·46	0·94	0·39	1·08	178
	78	59	42	—	6	4	21·84	20·1	1·74	1·07	0·41	1·33	200
	52	59	43	—	3	4	22·05	20·4	1·65	0·97	0·41	1·24	215
	30	36	20	1 (I)	2	4	25·23	23·4	1·83	1·13	0·43	1·41	296
V. Screen workers	123	43	21	—	½	1	5·44	4·3	1·14	0·65	0·31	0·82	116
	124	55	25	—	7	1	10·87	10·3	0·57	0·34	0·14	0·43	121
	69	66	—	—	?	2	13·93	12·3	1·68	1·04	0·43	1·25	109
VI. Unclassified underground	70	55	40	—	1	1	1·26	1·00	0·26	0·16	0·07	0·19	67
	15	59	—	—	—	1	1·28	1·1	0·18	0·11	0·08	0·10	139
	73	59	30	—	5	1	9·16	7·9	1·26	0·80	0·36	0·90	140
	20	44	—	—	—	2	2·14	1·6	0·54	0·34	0·16	0·38	154
	33	37	17	—	5	2	3·25	2·2	1·02	0·58	0·24	0·78	159
	21	77	51	—	12	2	4·44	3·8	0·64	0·41	0·18	0·46	151
	28	42	—	—	—	2	4·85	3·7	1·19	0·78	0·38	0·81	228
	12	53	35	5 (V)	0	2	7·47	6·3	1·17	0·74	0·32	0·85	117
	43	29	9	—	6	2	9·20	7·7	1·50	0·96	0·45	1·04	77
	48	65	39	—	5½	2	9·69	7·5	2·19	1·16	0·51	1·67	176
	32	66	45	—	?	3	2·52	2·0	0·52	0·29	0·13	0·39	169
	90	59	39	—	2	3	4·45	3·8	0·65	0·44	0·24	0·41	137
	67	59	46	—	0	3	4·50	2·8	1·70	1·10	0·50	1·20	104
	58	25	11	—	0	3	8·29	8·0	0·29	0·19	0·08	0·21	123
	100	67	—	—	—	3	12·22	9·2	3·02	1·80	0·75	2·28	128
	42	73	—	—	8	3	13·40	10·7	2·75	1·72	0·74	2·00	113
	81	74	—	—	9	4	9·88	5·4	4·48	2·82	1·25	3·22	166
	76	54	40	—	0	4	10·06	8·3	1·76	1·18	0·56	1·20	92

continued

Table 3 continued

Chief Occupation	P.M. No.	Age	Duration of Exposure (years)*			Pathological Grade†	Percentage Dry Lung						Dry Weight of Lung (g.)
			1	2	3		Total Dust	Coal	Siliceous Minerals	Silica	Quartz	Kaolin plus Mica	
VII. Tin-miners and granite-workers	40	—	4?	—	?	1	1·34	1·2	0·14	0·08	0·04	0·10	131
	117	62	25	—	11	5	0·80	0·29	0·51	0·22	0·14	0·37	149
	113	53	23	—	12	5	1·02	0·34	0·68	0·40	0·23	0·45	75?
	38	—	28	—	20	5	1·24	0·82	0·42	0·26	0·14	0·28	113
	127	62	30	—	17	5	1·61	0·66	0·95	0·63	0·30	0·60	64
	129	46	20	—	10	5	1·61	0·56	1·05	0·78	0·39	0·66	156
	116	57	—	—	?	5	1·67	0·61	1·06	0·66	0·35	0·71	137
	131	60	20	—	20	5	1·74	1·09	0·65	0·33	0·18	0·47	163
	119	61	34	—	10	5	1·86	0·55	1·31	0·77	0·33	0·98	102
	111	—	30	—	?	5	1·98	0·70	1·28	0·82	0·38	0·90	98
	109	51	34	—	?	5	2·01	0·73	1·28	0·96	0·46	0·82	62
	112	59	20	—	10	5	2·06	0·51	1·55	1·04	0·52	1·03	141
	118	58	20	—	18	5	2·91	1·77	1·14	0·73	0·34	0·80	114
	126	53	23	—	8	5	2·94	0·98	1·96	1·20	0·64	1·32	63
	36	—	12	2(VI)	14	5	3·24	1·40	1·84	1·33	0·57	1·27	87
VIII. Non-miners	13	50				0	0·68	0·6	0·08	0·031	ND	ND	155
	31	40				0	0·80	0·7	0·10	0·024	ND	ND	100
	88	30				0	0·82	0·33	0·49	0·26	ND	ND	110

ND = not determined.

* Duration of exposure: 1 = years spent in stated occupation; 2 = years spent in other underground occupation; 3 = years between removal from dust and death.

† Pathological grade: 1 = slight reticulation; 2 = reticulation; 3 = reticular and mixed nodulation; 4 = confluent fibrosis; 5 = silicotic nodulation.

TABLE 4

LUNG DUST COMPOSITION FOR DIFFERENT OCCUPATIONAL GROUPS FOR NEW AND OLD SERIES

Occupational Group	New Series (A)				Old Series (B)			
	No. of Men	Total Dust in Lungs (%)			No. of Men	Total Dust in Lungs (%)		
		Coal	Quartz	Kaolin plus Mica		Coal	Quartz	Kaolin plus Mica
Tin-miners	15	44	18	38*	—	—	—	—
Rock-workers	9	57	12	31	5	20	18	62
Hauliers, riders, etc.	10	70	10	20	4	84	3	13
Unclassified underground	18	79	6	15	—	—	—	—
Colliers	28	89	3	8	33	86	2	12

* Includes also felspar.

general atmospheric pollution. Likewise, the "coal" in the three normals probably represents atmospheric pollution. The average value of the three "normals", 1·3 g. of coal in both lungs, is slightly higher than the mean value for carbon in lungs of males recently given by Blacklock, Kennaway, Lewis, and Urquhart (1954) as 0·8 g.

As one proceeds from tin-miners to rock-workers in coal-mines, hauliers, and finally colliers, the dust becomes richer in coal and decreases in siliceous minerals; similar results obtained in the old series are shown in (B) Table 4. In the new series the proportions of quartz to kaolin plus mica decrease slightly in the same order of occupations, but the differences are very small.

Thus the quartz percentages of the siliceous minerals in the lung residues are:

Tin-miners	32
Rock-workers in coal-mines	28
Colliers	27

The dust as per cent. dry lung for different occupational groups is shown in Table 5 for total dust, coal, quartz, kaolin plus mica, and total silica. Here the differences between occupations are much more pronounced. Coalworkers' lungs as a class contain far more dust than tin-miners' or granite-workers' lungs, but this difference is mainly due to coal itself. The proportions of quartz and silicates are about the same, except that the rock-workers in coal-mines have significantly more quartz and silicates than all other groups. It is worth while pointing out here that the quartz and silicate contents of the lungs of colliers are not significantly (at the 10% level) different from those of tin-miners. This will be discussed further.

TABLE 5
LUNG DUST CONCENTRATION (% DRY LUNG) FOR DIFFERENT OCCUPATIONAL GROUPS

Occupation	No. of Men	Total Dust (%)	Coal (%)	Quartz (%)	Kaolin plus Mica (%)	Total Silica (%)
Tin-miners	15	1·87 ± 0·18*	0·81 ± 0·11	0·33 ± 0·04	0·72 ± 0·09†	0·68 ± 0·09
Rock-workers	9	6·58 ± 1·13	3·73 ± 0·65	0·80 ± 0·15	2·05 ± 0·44	1·80 ± 0·37
Hauliers, etc.	10	4·14 ± 0·77	2·91 ± 0·55	0·42 ± 0·185	0·80 ± 0·27	0·78 ± 0·28
Unclassified	18	6·56 ± 0·90	5·16 ± 0·73	0·39 ± 0·07	1·00 ± 0·20	0·87 ± 0·16
Colliers	28	10·95 ± 1·28	9·77 ± 1·23	0·30 ± 0·03	0·88 ± 0·11	0·72 ± 0·08

* Standard error of mean. † Includes also felspar.

TABLE 6
ESTIMATED AMOUNTS OF TOTAL DUST AND VARIOUS CONSTITUENTS IN AVERAGE LUNGS OF DIFFERENT OCCUPATIONAL GROUPS

Occupation	No. of Men	Mean Dust Exposure (years) *	Total Dust (g.)	Coal (g.)	Quartz (g.)	Kaolin plus Mica (g.)	Total Silica (g.)
Tin-miners	15	23	4·0 ± 0·4†	1·8 ± 0·3	0·7 ± 0·1	1·5 ± 0·2‡	1·4 ± 0·2
Rock-workers	9	37	20·2 ± 4·9	11·1 ± 2·4	2·5 ± 0·6	6·6 ± 2·0	5·7 ± 1·6
Hauliers, etc.	10	38	10·6 ± 2·5	7·0 ± 1·6	1·3 ± 0·7	2·3 ± 0·9	2·2 ± 1·0
Unclassified	18	34	17·4 ± 2·4	13·5 ± 1·8	1·1 ± 0·2	2·8 ± 0·6	2·4 ± 0·5
Colliers	28	33	34·7 ± 6·2	31·1 ± 5·8	0·9 ± 0·1	2·6 ± 0·4	2·2 ± 0·3

* Sum of 1 and 2 in Table 3. † Standard error of mean. ‡ Contains also felspar.

Table 6 shows the estimated amount of total dust and its components present in both lungs. The figures and trends are similar to those in Table 5 but the spread of values is larger. Colliers' lungs contain on an average 35 g. of dust, nearly nine times as much as is found in tin-miners' lungs. It will also be seen that colliers' lungs contain more quartz than the tin-miners' lungs (Fig. 8) although the difference is not statistically significant at the 10% level.

It should be noted that the lungs from Cornwall had been kept in formalin whereas those from South Wales were obtained fresh. It is possible that small amounts of dust may have been lost from the Cornish lungs, but such losses probably did not exceed 5% of the total dust.

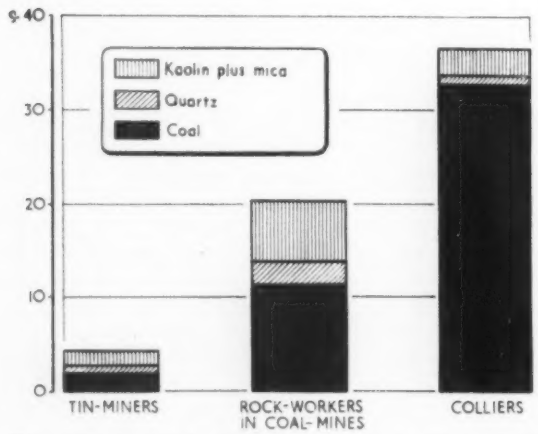

FIG. 8.—Amounts of dust constituents in average lungs of different occupational groups.

Effect of Pathological Grade.—As mentioned in the introduction, the following pathological classification was used in conformity with the previous work.

Grade No.	Description	Approximate Radiographic Equivalent
1	Slight reticulation	Simple pneumoconiosis
2	Reticulation	
3	Mixed nodulation	
4	Confluent fibrosis	Progressive massive fibrosis
5	Silicotic nodulation	

Grade 3 corresponds mainly to simple pneumoconiosis, but may also contain cases with " infective" nodules.

This classification is mainly based on the type of lesion revealed by study of macro- and micro-sections and not necessarily on the amount of fibrotic tissue. The radiographic equivalents are based on the data given in Table 12.

Taking all cases together, Table 7 shows the mean values for coal, quartz, silicates, and also total silica as percentages of dry lung. There is a regular increase from grade 1 to 4 although the differences between grade 3 and 4 and also the increase in coal content from grade 1 to 2 are not statistically significant at the 10% level. The actual significance levels of the differences are listed in Table 7a. If a difference is statistically significant at, say, the 1% level, it means that there is less than one chance in a hundred that such a difference could have arisen due to chance alone.

TABLE 7

DUST CONCENTRATION IN RELATION TO PATHOLOGICAL GRADE FOR ALL CASES

Pathological Grade	No. of Men	Mean Dust Exposure (years)*	Percentage Dry Lung				
			Total Dust (%)	Coal (%)	Quartz (%)	Kaolin plus Mica (%)	Total Silica (%)
1	12	26	3·85	3·31	0·15	0·38	0·33
2	20	33	5·75	4·69	0·30	0·77	0·66
3	20	38	9·70	8·06	0·45	1·18	1·02
4	19	37	12·98	10·89	0·53	1·55	1·30
5	15	24	2·17	0·83	0·46	0·88	0·86

* Sum of 1 and 2 in Table 3.

TABLE 7a

SIGNIFICANCE LEVELS OF DIFFERENCES OF MEAN VALUES GIVEN IN TABLE 7

Difference of Pathological Grades	Significance Level (%) for				
	Total Dust	Coal	Quartz	Kaolin plus Mica	Silica
2–1	NS	NS	5	5	5
3–2	1	2	5	5	5
4–3	NS	NS	NS	NS	NS
4–2	0·1	1	5	1	2

NS = not significant at the 10% level.

A co-variance analysis was carried out to investigate the effect of exposure time on the quartz and coal dust content of the lungs. As the exposure times did not have a sufficiently large range, the exposure effect was not significant at the 10% level. The analysis did, however, confirm that in general there are significant differences between grades. It is quite obvious that grade 5, the silicotic nodulation, does not fit into the picture of regular increase of dust concentration with increasing grade of pathology.

The work described in this report had been undertaken in continuation of the previous analyses in the hope that the data could be pooled and thus more significant results be obtained. On comparing the average results (percentages of dry lung) for the different pathological groups it was found, however, that the new results were in general considerably lower than the old ones. The data for all cases, namely 54 lungs in the old and 87 in the new series, are assembled in Table 8 and for the more homogeneous groups of colliers (27 in each series) in Table 9.

The differences between the two series are probably due to selection of lung material. For the old series representative fibrotic areas of the lungs had been chosen and it appears very likely that lower dust percentages would have been found had a whole lung been analysed as was done in the new series. There is, however, another difference between the two series. The lungs in the old series came chiefly from anthracite-workers, whereas in the new series they came chiefly from steam-coal workers. It appears to us very unlikely that this should account for lower dust proportions at a given pathological stage, as it would make a unit of steam-coal dust more pathogenic than a unit of anthracite dust and no trace of such an effect had been found in the old series ; nevertheless it points to a gap in our knowledge.

It is intended to remedy this deficiency by analysing, in the future, more lungs from anthracite and bituminous areas of South Wales as well as from other English and Scottish coalfields. For the present it was felt better, however, to restrict a detailed evaluation to the results of the new series.

Ignoring for the moment the differences in level of dust concentrations between the old and new series, it will be seen from Tables 8 and 9 that the trends for increasing proportions of dust with increasing grade of pathology are similar in both series for the grades 1 to 4. Grade 5 is not comparable at all. In the old series three rock-workers in coal-mines with very high silica content of the lungs were classed as grade 5, whereas in the new series there is only one repairer accompanied by 15 tin-miners and granite-workers in this grade (classical silicosis).

TABLE 8

DUST CONCENTRATION IN RELATION TO PATHOLOGICAL GRADE FOR ALL CASES AND MEAN VALUES OF DUST FOR THE OLD AND NEW SERIES

Pathological Grade	No. of Lungs		Exposure (years)		Percentage Dry Lung									
					Total Dust		Coal		Quartz		Kaolin plus Mica		Silica	
	Old	New	Old	New	Old	New	Old	New	Old	New	Old	New	Old	New
1	—	12	—	26	—	3·85	—	3·31	—	0·15	—	0·38	—	0·33
2	14	20	38	33	12·47	*5·75*	10·71	*4·69*	0·28	0·30	1·48	**0·77**	0·94	*0·66*
3	13	20	38	38	13·79	*9·70*	11·42	*8·06*	0·34	0·45	2·03	**1·18**	1·25	1·02
4	24	19	31	37	16·52	12·98	14·25	10·89	0·37	*0·53*	1·90	1·55	1·23	1·30
5	3	15	18	24	4·76	*2·17*	0·73	0·83	0·96	*0·46*	3·07	**0·88**	2·34	*0·86*

Italicized values are different at 10% significance level comparing old and new series. Bold type values are different at 1% significance level comparing old and new series.

TABLE 9

DUST CONCENTRATION IN RELATION TO PATHOLOGICAL GRADE FOR COLLIERS ONLY AND MEAN VALUES OF DUST FOR THE OLD AND NEW SERIES

Pathological Grade	No. of Lungs		Exposure (years)		Percentage Dry Lung									
					Total Dust		Coal		Quartz		Kaolin plus Mica		Silica	
	Old	New	Old	New	Old	New	Old	New	Old	New	Old	New	Old	New
2	8	8	41	30·9	14·59	**5·66**	12·78	**4·87**	0·25	0·22	1·57	**0·57**	0·95	*0·49*
3	7	9	36	33·0	14·65	12·36	12·30	11·06	0·32	0·32	2·03	**0·97**	1·23	**0·79**
4	12	10	35	36·2	18·12	14·74	16·06	13·26	0·31	0·36	1·74	*1·12*	1·10	0·91

Italicized values are different at 10% at least significance level comparing old and new series. Bold type values are different at 1% at least significance level comparing old and new series.

TABLE 10

ESTIMATED AVERAGE AMOUNT OF TOTAL DUST AND VARIOUS CONSTITUENTS IN BOTH LUNGS OF MEN FROM DIFFERENT PATHOLOGICAL GRADES FOR ALL CASES

Pathological Grade	No. of Men	Mean Dust Exposure (years)*	Total Dust (g.)	Coal (g.)	Quartz (g.)	Kaolin plus Mica (g.)	Total Silica (g.)
1	12	26	9·1	7·9	0·35	0·86	0·76
2	20	33	15·3	12·4	0·80	2·06	1·77
3	20	38	27·3	23·0	1·19	3·15	2·71
4	19	37	47·7	40·3	1·88	5·53	4·60
5	15	24	5·2	1·9	1·15	2·09	2·05

* Sum of 1 and 2 in Table 3.

TABLE 10a

SIGNIFICANCE LEVELS OF DIFFERENCES OF MEAN VALUES GIVEN IN TABLE 10

Difference of Pathological Grades	Significance Level (%) for				
	Total Dust	Coal	Quartz	Kaolin plus Mica	Silica
2–1	10	10	5	5	2
3–2	1	1	5	5	5
4–3	5	10	10	5	5
4–2	0·1	1	1	1	1

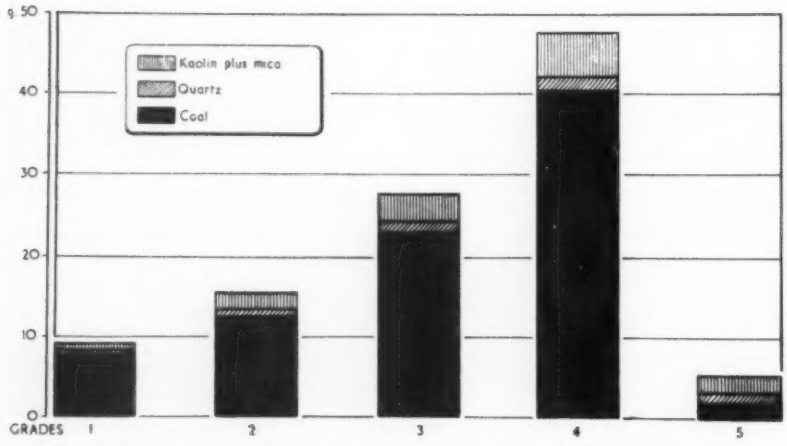

FIG. 9.—Total dust in lungs for different pathological grades (all cases).

Mean values of total dust in lungs for the different pathological groups are shown in Table 10 and Fig. 9. The Table shows the same trends as Table 7, but in a more pronounced form, because the lungs tend to become heavier with increasing pathological grade. Nevertheless, even on the basis of total dust, quartz, or silica the increase from grade 3 to grade 4 is not statistically significant ; this is due to the large variations found within each pathological group and especially in grade 4. Table 11 and Fig. 10 show similar data for the more homogeneous group of colliers.

The increase in lung weight with increasing pathological grading is significant (at the 1% level) and is not entirely accounted for by the dust itself, as may be seen from the following figures :—

Grade 4 lungs are on an average 20 g. heavier than those of grades 2 and 3. This increase in weight is probably mainly due to fibrotic tissue and the main constituent of such tissue is collagen. A technique of collagen determination, recently developed for use in animal experiments (Stacy and King, 1954) was used on average samples of dried lung from the different pathological grades. The following results were obtained :—

The difference in collagen between grade 4 and the average of grades 1, 2, and 3 is of the order of 6 g.

Dense fibrotic lung tissue of rats produced in experimental silicosis was found to contain 40% of collagen. Using this value, 6 g. of collagen correspond to 15 g. of fibrotic tissue, in reasonable agreement with the 20 g. increase in weight of dust-free lungs of grade 4 over those of grades 1, 2, and 3.

TABLE 11

ESTIMATED AVERAGE AMOUNT OF TOTAL DUST AND VARIOUS CONSTITUENTS IN BOTH LUNGS OF MEN FROM DIFFERENT PATHOLOGICAL GRADES FOR COLLIERS ONLY

Pathological Grade	No. of Men	Dust Exposure (years)	Total Dust (g.)	Coal (g.)	Quartz (g.)	Kaolin plus Mica (g.)	Total Silica (g.)
1	1	16	5·2	4·9	0·08	0·26	0·22
2	8	31	15·5	13·5	0·58	1·45	1·28
3	9	33	38·1	34·0	1·00	3·04	2·46
4	10	36	54·2	49·2	1·21	3·78	3·06

TABLE 11a

SIGNIFICANCE LEVELS OF DIFFERENCES OF MEAN VALUES GIVEN IN TABLE 11

Difference of Pathological Grades	Significance Level (%) for				
	Total Dust	Coal	Quartz	Kaolin plus Mica	Silica
3–2	1	1	2	1	1
4–3	NS	NS	NS	NS	NS
4–2	5	5	5	5	5

NS = not significant at the 10% level.

TABLE 12

WEIGHTS OF DUST AND LUNGS FOR DIFFERENT PATHOLOGICAL GRADES

Pathological Grade	Mean Dry Weight of Right Lung (g.)	Mean Total Dust in Right Lung (g.)	Mean Weight of Right Lung Minus Dust (g.)
0	111	1·1	110
1	113·3	4·6	108·7
2	136·8	7·6	129·2
3	140·1	13·7	126·4
4	171·3	24·0	147·3
5	113·1	2·2	110·9

TABLE 13

COLLAGEN IN LUNGS OF DIFFERENT PATHOLOGICAL GRADES

Pathological Grade	Collagen in Dried Lung (%)	Collagen in Dust-free Right Lung (g.)
0	5·0	5·5
1	5·6	6·1
2	4·5	5·8
3	5·2	6·6
4	8·3	12·2
5	7·5	8·3

Radiography.—A comparison of Table 3 with the similar data given for the old series (Table 1 in King and Nagelschmidt, 1945) will show that *x*-ray data have been omitted, although *x*-ray assessments were available for some but not all the men in the new series. These radiographs had been taken between 1944 and 1947 under conditions which would not now be regarded as satisfactory. An attempt has been made to read these old radiographs by experienced radiologists of the Pneumoconiosis Research Unit, Cardiff, and the results are given in Table 14. They show a general parallelism between amount of dust, pathological grading, and radiological reading, and they also show that, with one exception, the pathological grades 1 to 3 used in this paper correspond to radiological diagnosis of simple pneumoconiosis and grade 4 to progressive massive fibrosis as might be expected. But the information is too scanty to be analysed in detail. It is hoped in future work to make more detailed comparison between satisfactory radiographs, a new pathological classification, and detailed lung dust analyses.

Size of Lung Dust.—Some preliminary work on measurement of the particle size distribution of the lung residues has been done (Cartwright and Nagelschmidt, 1951). The techniques developed, using light microscopy only, were not entirely satisfactory and further study by light and electron microscopy is required. Trypsin digestion was found to be the least destructive method of isolating the lung dust, and size distributions of the mean of three lung dusts are shown in Fig. 6. The size of the coal is generally larger than the size of the non-coal. This has recently been confirmed by electron microscope observations (Policard and Collet, 1953).

Discussion

In the preceding sections the analytical data for the dust found in the lungs of 71 pneumoconiotic workers from South Wales and of 15 silicotic tin-miners and granite-workers from Cornwall have been presented. It remains to be seen what information can be derived from the results which might throw

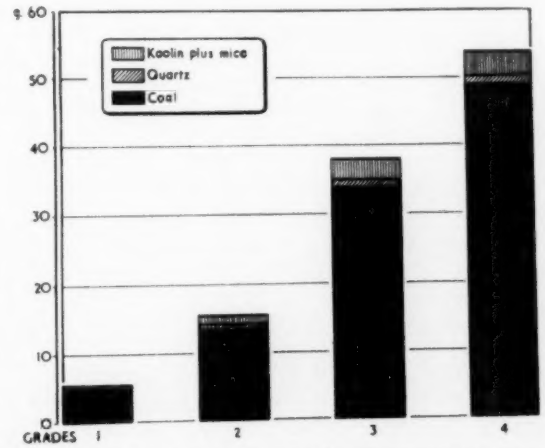

FIG. 10.—Total dust in lungs for different pathological grades (colliers only).

TABLE 14

RADIOLOGICAL ASSESSMENT OF SELECTED LUNGS IN RELATION TO PATHOLOGICAL GRADE AND TOTAL DUST

Necropsy No.	Pathological Grade	X-ray Reading (P.R.U. Classification)	Total Dust (% Dry Lung)
70	1	Normal	1·2
92	1	"	1·8
62	1	"	3·7
63	2	"	1·1
20	2	1	2·1
91	2	Normal	3·5
17	2	"	4·1
27	2	*1	4·8
2	2	*Normal	5·4
47	2	*1	5·7
53	2	*1	6·6
12	2	*1	7·5
48	2	2	9·7
86	2	1	12·5
90	3	1	4·4
7	3	3 B	7·2
57	3	*2	8·2
4	3	*2	8·3
44	3	*1	4·8
95	3	3	17·4
93	4	P.M.F. B	3·2
107	4	P.M.F.	4·4
96	4	P.M.F. D	6·5
72	4	P.M.F. D	8·3
77	4	P.M.F. C	13·6
9	4	P.M.F. B	14·0
10	4	P.M.F. B	19·8
78	4	P.M.F. C	21·8
52	4	P.M.F. B	22·0
59	4	P.M.F. C	31·4

*Assessment doubtful in view of poor quality of film.

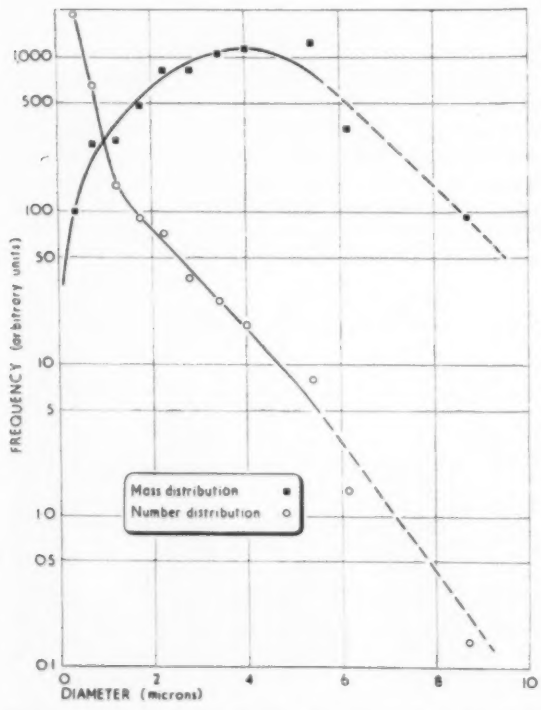

FIG. 6.—Number and mass distribution of trypsin digested residues.

light on the aetiology and method of progression of the disease. It should be borne in mind that the sample of lungs was obtained from post-mortem material from March, 1946, to March, 1947. The lungs were collected until equal numbers of different pathological grades were obtained, but the cases used were not necessarily consecutive ones in any one grade, as collection had to be adjusted to the available facilities for drying the lungs, and in general only those cases were used where senior staff had carried out the necropsy. Of all cases occurring in the period of one year, roughly half were used for grade 1, half for grade 2, about two-thirds for grade 3, and a little more than one-fifth for grade 4.

The assessment of the pathological grade is considered to be entirely reliable for grade 4 and to have a very small observer error for the other grades (Gough, private communication).

Statistically the source of data is not completely satisfactory. It would have been better if the cases could have been selected at random out of those available in the period of a year. However, this was not possible for the reasons stated. Nevertheless, it was assumed in analysing the data that the cases used were a random sample from the population of post-mortem cases.

A great difficulty in interpreting the results is the large variability of the data even within given occupational and pathological groups. For instance, colliers with fibrosis classed as grade 4 ranged in age from 36 to 64 years, and in working time from 21 to 43 years' dust exposure. One man collected 150 g. of dust in 21 years of dust exposure, another 20 g. in over 40 years, while a third collected 70 g. of dust in 40 years. Similar examples can be given for grade 2 colliers where the pneumoconiosis was probably not the cause of death. The causes of this variability are probably external variations in dust concentrations and internal variations in individual response. It also appears that the pathological classification used is too simple. It refers to the type of lesion and the maximal alteration of the lung tissue, but it does not consider the amount of lesions throughout the lung. A more elaborate classification based on type and amount of fibrosis would probably result in a better fit with the dust analyses.

The following discussion can be divided into two parts : (1) The relative hazard of different occupations, and (2) the relative hazard of different dust constituents.

Relative Hazard of Different Occupations.—A rough estimate of the relative hazard of different occupations can be obtained from the working histories and the mean pathological grading without

reference to the lung dust analyses. The results are shown in Table 15 for those occupation groups which have a reasonable number of cases, and also for anthracite colliers and rock-workers from the old series.

TABLE 15

MEAN PATHOLOGICAL GRADE AND AVERAGE DUST EXPOSURE YEARS FOR DIFFERENT OCCUPATIONAL GROUPS

Occupation	Series	No.	Mean Pathological Grade	Exposure (years)
Tin and granite ..	New	15	4·7	23
Rock-workers* ..	Old	5	4·6	22
Rock-workers* ..	New	9	3·1	37
Colliers	Old	27	3·1	39
Colliers	New	28	3·0	33
Hauliers, etc. ..	New	10	2·1	38

*In coal-mines.

It will be seen that the tin- and granite-workers and the rock-workers of the old series who mostly had silicosis (grade 5) after 22 and 26 years form a first group with the highest hazard.

The colliers and the rock-workers of the new series took about 10 years more (33 to 39 years) to reach grade 3 and form a second group. The hauliers, who took on an average 38 years to reach grade 2, form a third group with the smallest hazard. As Table 3 shows, these men had also been engaged for variable times as rock-workers or colliers. It is noteworthy that there is only one collier with pathological grade 1 in the series. As equal numbers of the different pathological grades were collected and as colliers are the predominating occupational group, a higher number might have been expected.

The difference between hauliers and colliers may reflect a difference in dust concentration ; this is in general higher at the coal-face than in the roadways. Hart and Aslett (1942) concluded from radiological surveys that the occupation of haulier and of other men working in the roadways had a much smaller pneumoconiosis risk than the work of the colliers. The difference between colliers and tin-miners appears to be due to a different type of disease, silicosis developing comparatively faster than pneumoconiosis of coalworkers.

The mean duration of dust exposure for different pathological grades is given in Table 16.

To what extent the figures represent a progression of disease, in the sense that it takes 26 years to reach grade 1 and another 11 or 12 years to reach grade 3 or 4, cannot be said from these data. Radiological evidence given by Fletcher (1955), however, indicates a positive relationship between simple pneumoconiosis and dosage of dust.

TABLE 16

MEAN EXPOSURE YEARS FOR DIFFERENT GROUPS

Occupation	Pathological Grade			
	1	2	3	4
New series all cases.. ..	26 (11)*	33 (17)	38 (18)	37 (18)
New series colliers only ..	—	31 (8)	33 (9)	36 (10)

*The figures in parentheses give the number of men in each grade.

By combining the exposure years and lung analyses one can calculate the mean amounts of dust retained per year. The results for coal, quartz, and total dust are shown in Table 17 for the main occupational groups of the new series and separately for the different pathological grades amongst the colliers. The average collier in our series retained about 1 g. of dust per year (averaged over 33 years).

TABLE 17

MEAN RATE OF DUST ACCUMULATION FOR DIFFERENT OCCUPATIONAL GROUPS AND GRADES OF PATHOLOGY

Occupational Group	No. of Men	Pathological Grade	Dust Retained per Year (mg.)		
			Total Dust	Coal	Quartz
Tin-miners	15	—	180	80	32
Rock-workers in coal-mines ..	9	—	550	300	67
Hauliers	10	—	280	190	34
Colliers	28	—	1,050	950	27
Colliers	8	2	500	435	19
Colliers	9	3	1,155	1,030	30
Colliers	10	4	1,506	1,367	34

It may be asked how much dust is needed, in terms of particles per cc. between 1 and 5 microns to yield 1 g. of dust per year, which is the amount retained by the average collier. Assuming a breathing rate of 30 litres per minute and 300 five-hour shifts per year, the man would breathe 2,700 m^3. per year, or, say 3,000 m^3. while underground. Thus he would have to retain between 0·3 and 0·4 mg. of dust per m^3. of air breathed. Assuming an exponential size distribution (Wynn and Dawes, 1951), 1,000 particles per cc. of size 1 to 5 microns correspond to about 5·4 mg. of dust per m^3.

If all airborne dust were retained a concentration of 50 to 70 particles per cc. would give 1 g. per year. Using the alveolar retention curve of Brown, Cook, Ney, and Hatch (1950) the dust concentration to yield 1 g. in the lungs is approximately four times higher, leading to 200 to 300 particles per cc. of size 1 to 5 microns. This is lower than the average dust concentration found now in South Wales coal-mines and probably much lower than it had been in the days before dust suppression was practised, when the workers accumulated the dust in their lungs. But, Brown and others (1950) measured the instan-

taneous dust retention. What is not known at present is the relation between the amount temporarily retained in the alveolar tissue and the amount permanently retained. Therefore it is not possible to estimate the average dust content of the air to which the men had been exposed from the amount of dust found in their lungs.

Relative Hazard of Different Dust Constituents.—It is generally agreed that pneumoconiosis of coalworkers is due to dust, but there are two schools of thought about the importance of quartz. In the German and American literature it is generally stated that quartz is the causative agent, and that the disease is a modified silicosis which looks different in the lungs on account of the large amounts of coal which happen to accompany the quartz. In the British literature, on the other hand, the opinion is expressed that it is the coal dust itself which mechanically by its mere presence causes the series of lung changes found in simple pneumoconiosis, but that an additional factor, possibly infective, is necessary for the initiation of P.M.F.

The fact that quartz can cause silicosis is not in doubt. Whether a completely quartz-free coal dust can cause disabling pneumoconiosis is not at present known as dusts from coal-mines are never sufficiently low in quartz to establish this point. Even coal trimmers were found to have abnormal amounts of quartz in their lungs. The best evidence could be obtained from dust exposures to carbon black or synthetic graphite if the exposures were sufficiently high and workers who worked in such dust for a sufficient length of time could be found. Gärtner and Brauss (1951) have recently examined soot-workers and found radiological abnormalities classed as grade 2 and 3 pneumoconiosis after 15 or more years' exposure to carbon black dust but they found little or no disability. This is a matter for further survey work of great importance which should be undertaken. Rüttner, Bovet, and Aufdermaur (1952) have described a case of lung fibrosis in a graphite worker, where graphite and carborundum but no quartz at all were found in the lung dust.

It is, however, not necessary to make a decision between the two theories. It appears to us possible that both excessive amounts of coal and the quartz in the dusts contribute to the disease, and our data give some information about their relative proportions. The average amounts of coal and quartz and the coal/quartz ratio for all lungs from South Wales are shown in Table 18.

The quartz in the lungs comes chiefly from rock dust and the values are useful in giving a rough idea of the relative importance of coal- and rock-dust, especially if the rock is rich in quartz. It is notable that the proportions of coal to quartz are approximately the same in categories 1, 2, and 3 (simple pneumoconiosis) as in category 4 (P.M.F.). This gives no support to the theory that quartz is an important factor in the aetiology of P.M.F. and agrees with Cochrane's (1955) finding that in the Rhondda Fach there was no difference in the exposure to stone dust of men with simple pneumoconiosis as compared with those with P.M.F. The findings support the view (Gough, 1947 ; Heppleston, 1951 ; James, 1954) that massive pneumoconiosis is due to a second factor, usually tuberculosis, in addition to the action of the dust. Category 4 cases are all of massive fibrosis whereas there are none of these in category 3, yet the amount and character of the dust is not significantly different in the two groups. Thus, in these two groups, while the dust is similar, pathological changes are very different.

TABLE 18
RATIO OF COAL TO QUARTZ IN THE LUNGS FOR DIFFERENT GRADES OF PATHOLOGY

Pathological Grade	No. of Men in Group	Dust in Lungs (g.)		Coal/Quartz Ratio
		Coal	Quartz	
1	12	7·9	0·35	22·6
2	20	12·4	0·80	15·5
3	20	23·0	1·19	19·3
4	19	40·3	1·88	21·4
5	15	1·9	1·15	1·6

The action of coal and quartz on human lungs is not necessarily simply additive. Certain evidence from animal experiments suggests a more complicated mechanism. It has been shown (Ray, King, and Harrison, 1951) that very small amounts of quartz injected into the lungs of rats are eliminated from the lungs into the lymph nodes and therefore cause less lung fibrosis when they are injected alone than when they are injected with large amounts of coal which prevent this loss from the lung. From the evidence produced in this paper, however, it appears that coal may have an inhibiting effect on the fibrogenic action of quartz, possibly by simple dilution, since the amounts of quartz in the lungs of Cornish tin-miners with silicosis were very similar to those in coal-miners with simple pneumoconiosis, and the accompanying, possibly antidotal, kaolin and mica were only in slightly lower ratio to the quartz in the silicotics than in simple pneumoconiotics.

The lung dust analyses have shown that all classes of coalworkers including the colliers have more quartz in their lungs than the silicotic tin-miners and granite-workers (Table 6). If the different pathological grades of colliers are separated this holds for grades 3 and 4 and only colliers of

grade 2 have slightly less. It is true for the total quartz in the lungs and for the calculated annual increments shown in Table 17. But we cannot say from our findings whether the coal, kaolin, or mica is inhibiting the effect of the quartz or whether they are themselves responsible for the fibrosis and could have caused it in the absence of quartz.

Summary

In continuation of previous work lung dust analyses were made of 71 pneumoconiotic coal-workers from South Wales and of 15 silicotic tin-miners and granite-workers from Cornwall. In classifying the material five pathological grades and seven occupational groups were distinguished.

Coal, total silica, quartz, and the sum of kaolin and mica were determined by chemical and *x*-ray diffraction methods and the amounts of these constituents and of the total dust in the lungs were estimated.

The dust composition was found to vary for different occupational groups. The chief characteristic was the percentage of coal in the dust which fell in going from colliers to hauliers, rock-workers, and tin- and granite-workers. The quartz percentage of the siliceous minerals rose slightly in the same order of occupations. "Coal" in tin-miners' lungs is probably chiefly soot from acetylene lamps or candles and atmospheric pollution.

The amount of dust in the lungs increased with the pathological grade but differences between grades 3 and 4 were not significant. This lends support to the view that a second factor is necessary to account for the difference. On an average the colliers' lungs contained 35 g. of total dust of which 0·9 g. was quartz. The tin-miners had 4 g. of total dust and 0·7 g. of quartz. The calculated average dust accumulation rate for colliers was of the order of 1 g. of dust per year, but it is doubtful whether this rate is constant.

Although the quartz percentage of the lung dust was much higher in silicotic tin-miners than in colliers with pneumoconiosis, the absolute amounts of quartz in their lungs were similar. Rock-workers in coal-mines had over three times more quartz in their lungs than the silicotic tin-miners.

The proportion of coal to quartz for the whole group of lungs from South Wales was of the order of 20 : 1. It appears likely that both total dust and quartz contribute to the pneumoconiosis of coal-workers.

We are grateful to Professor J. Gough who provided the lungs from South Wales, made the pathological assessments, and secured the working histories, and to Dr. L. W. Hale, who provided the lungs from Cornwall and their working histories. A detailed pathological description of the material will be published by Professor J. Gough. Miss V. Pash and Mr. W. Weeden gave help with the analyses and Miss A. V. Sanderson and Mrs. C. Wrightam with the computations. We are also grateful to Dr. J. C. Gilson and the staff of the Pneumoconiosis Research Unit for *x*-ray readings and helpful criticism, to Dr. J. O. Irwin for statistical advice, and to the Medical Research Council for financial assistance. Most of the *x*-ray diffraction analyses were made at the National Physical Laboratory. Figs. 6 to 10 are reproduced by permission of the Controller, H.M. Stationery Office.

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CARDIAC HYPERTROPHY IN COALWORKERS' PNEUMOCONIOSIS

BY

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Pneumoconiosis of coalworkers is a pulmonary disease which can produce a striking effect on the heart (Thomas, 1948; Lavenne, 1951; Wells, 1954a). Many coalworkers in the southern part of Wales develop the pulmonary lesions. In some there are numerous foci of accumulated dust scattered more or less uniformly throughout each lung; the foci are discrete, usually less than 7 mm. in diameter, and contain little or no collagenous fibrous tissue. This condition is called simple pneumoconiosis. There may be areas of focal emphysema in and around the dust foci (Heppleston, 1953). The lungs of other miners show in addition to simple pneumoconiosis one or more dust-pigmented fibrous lesions. Initially, these fibrous areas are less than 1 cm. across but slowly enlarge and in the later stages more than half of each lung may be destroyed by the fibrosis. This condition is referred to as progressive massive fibrosis or massive pneumoconiosis. It is often complicated by bullous or diffuse emphysema (Fletcher and Gough, 1950).

The presence of pulmonary heart disease becomes obvious when right heart failure appears but this is a late and often terminal event, and it was of interest to assess earlier stages of pulmonary heart disease in relation to the extent of the pneumoconiosis. This work therefore compares the right ventricle studied at necropsy with the pulmonary disease, and relates the electrocardiogram to the necropsy findings in some cases.

The electrocardiographic signs of pulmonary heart disease used in this study have been the vertical heart pattern or right axis deviation with well developed pointed P in leads II and III, T inversion in lead III or downward inclination of the ST segment in II and III, a qR pattern in VR, a deep S wave in V6 and R greater than S in V1, or M-shaped QRS complex in V1. The unusual standard lead pattern of prominent S_I, S_{II}, and S_{III} has also been accepted in conjunction with the other signs. Not all the above signs were present in every case, but most of them had to be present before the electrocardiogram was regarded as indicative of pulmonary heart disease.

Routine necropsies have been used for part of this work and in these a thickness of 5 mm. or more of the right ventricular wall has indicated hypertrophy. A selected number of hearts have been dissected and the ventricles weighed using the method of Herrmann and Wilson (1922). By this method the normal range of the ratio of left ventricle to right ventricle (LV/RV ratio) was 1·46 to 2·14. Thus ratios below 1·46 indicate right ventricular hypertrophy but it was found in practice that ratios below 1·00 were comparable to a thickness of 5 mm. or more, and this group is referred to as marked right ventricular hypertrophy. The intermediate group with an LV/RV ratio lying between 1·46 and 1 and not identified by thickness of the wall is referred to as early right ventricular hypertrophy.

All the material used in this study was of necessity selective but as the object was to study the relationship between cardiac hypertrophy and pneumoconiosis rather than the absolute incidence of the phenomenon the material was acceptable.

Results

The overall pattern is shown by a scrutiny of the protocols of the necropsies made on 1,000 coalworkers during the years 1947–50 at the Institute of Pathology of the Welsh National School of Medicine. These were cases coming to necropsy because of a history of exposure to coal dust but the causes of death were various. Of the 1,000 cases, 546 had simple pneumoconiosis and 454 had massive fibrosis. The latter was diagnosed if either lung contained a dust-pigmented fibrous lesion more than 3 cm. across.

Of the 546 cases with simple pneumoconiosis, only eight were considered at necropsy to have died

of pulmonary heart disease due to the simple dust disease alone. This opinion was based on a clinical history of congestive heart failure and naked-eye pathological evidence of severe simple pneumoconiosis with right ventricular hypertrophy and a lack of any other cause for the right cardiac enlargement. In a further seven cases with right ventricular hypertrophy death was not due to the simple pneumoconiosis but the latter appeared to be the only cause for the right ventricular change. Thus, on the basis of gross post-mortem appearances the series contained 15 cases in which the simple dust disease alone had affected the right heart. The lungs in these cases were subsequently studied microscopically and by the use of whole lung sections (Gough and Wentworth, 1949), and this investigation showed evidence in each case of chronic inflammation of the smaller bronchi and of slight degrees of diffuse emphysema. It was not possible to prove that the co-existing chronic bronchiolitis or emphysema was due to the simple pneumoconiosis. On the contrary, Gough (1952) has drawn attention to a centrilobular focal type of emphysema with bronchiolitis in lungs free of dust. Moreover, Leopold (1955) has shown that right ventricular hypertrophy may result from chronic bronchiolitis. It is, therefore, possible that in the 15 cases of simple pneumoconiosis described the right ventricular hypertrophy was due partly to bronchiolitis or emphysema. We had to conclude that simple pneumoconiosis alone occasionally caused right heart hypertrophy, but would suggest that chronic bronchiolitis also played a part.

The 454 cases of massive fibrosis included 412 with other co-existing lung lesions of various degrees. The common co-existing lesions were generalized emphysema, focal emphysema, chronic bronchiolitis, and pulmonary tuberculosis. In the 43 remaining cases the lungs showed massive pneumoconiosis only. The extent of the emphysema was judged by naked-eye examination of the fresh lungs and the whole lung sections. It was deemed to be insignificant if it was similar in extent to that seen in the lungs of individuals who have not had symptoms of emphysema. For example, the small emphysematous areas shown in Fig. 1 were deemed to be insignificant. The 43 cases were classified according to the size of the massive dust lesion. When the lungs contained more than one lesion the classification was based on the maximum diameter of the largest one. In nine the diameter was less than 5 cm., in 15 it was between 5 and 10 cm., and in 19 it was over 10 cm. The cases were also classified according to the presence or absence of right ventricular hypertrophy. The latter was deemed to be present if the maximum right ventricular thickness was over 5 mm. The 43 cases were then analysed to determine whether any relationship existed between the size of the lesions and the incidence of right ventricular hypertrophy. The analysis is displayed in Table 1.

FIG. 1.—Lung section (about half natural size) from a case in which progressive massive fibrosis with minimal emphysema had produced marked right ventricular hypertrophy.

TABLE 1
INCIDENCE OF RIGHT VENTRICULAR HYPERTROPHY ACCORDING TO SIZE OF LESION

	Less than 5 cm.	5–10 cm.	Over 10 cm.	Total
Right ventricular hypertrophy	1	13	17	31
Right ventricular hypertrophy absent	8	2	2	12
Totals	9	15	19	43

On studying the ages of these cases it was noted that the mean age of those with small lesions was 52 years, with moderately sized lesions 65 years, and with large lesions 60 years. It was possible, therefore, that increased age contributed somewhat to increased incidence of right ventricular hypertrophy, but the figures also show that the incidence of right ventricular hypertrophy increased with increasing size of the dust lesion in the cases of massive pneumoconiosis and minimal emphysema.

In simple pneumoconiosis right ventricular hypertrophy was an uncommon finding, 15 in 546 cases, and in these there was a degree of chronic bronchiolitis whose importance needs further investigation. Right ventricular hypertrophy was common in massive fibrosis. Cases of pure massive fibrosis were not common, 43 in 454 cases, but of the 43 there were 31 with marked right ventricular hypertrophy, suggesting a relationship between massive fibrosis alone and right ventricular hypertrophy.

Eighty hearts from necropsies on coalworkers were dissected and the ventricles weighed (Table 2).

TABLE 2

ANALYSIS OF VENTRICULAR WEIGHTS OF 80 PNEUMOCONIOTIC HEARTS

Stage of Disease	Marked Right Ventricular Hypertrophy (LV/RV ratio less than 1)	Early Right Ventricular Hypertrophy (LV/RV ratio 1·00 to 1·46)	Normal LV/RV Ratio above 1·46	Totals
Progressive massive fibrosis	32	19	5	56
Simple pneumoconiosis	4	11	9	24

Fifty-six cases had progressive massive fibrosis. Thirty-two of these had marked hypertrophy with an LV/RV ratio below 1, 19 were cases of early right hypertrophy with an LV/RV ratio between 1 and 1·46, and five were normal. Of the 19 patients with early right hypertrophy, eight died of conditions not directly related to pneumoconiosis and 11 died of heart failure due to pneumoconiosis and emphysema. It was apparent that massive fibrosis was associated with some degree of right ventricular hypertrophy in all but five of the 56 cases, but in the 19 with early right ventricular hypertrophy clinical evidence of pulmonary heart disease would be difficult to find before the onset of right heart failure.

There were 24 cases of simple pneumoconiosis four of which had a ratio below 1 indicating marked right ventricular hypertrophy. In three of these there was severe bullous emphysema and the fourth had had anaemia for a long period.

Early right ventricular hypertrophy (LV/RV ratio between 1·46 and 1) was present in 11 cases. In five of these emphysema of considerable degree was present, two had pulmonary tuberculosis, and another had severe chronic bronchitis. These factors may have been contributory causes of the early right ventricular hypertrophy. The three remaining cases of simple pneumoconiosis appeared to have this as the only explanation of the early right ventricular hypertrophy.

It was of interest that in five of the cases of early right ventricular hypertrophy with electrocardiograms available not one electrocardiogram had the signs of right ventricular hypertrophy but all five had pulmonary P waves and all but one had a vertical heart pattern.

This analysis suggested that the right ventricular hypertrophy found in cases of massive fibrosis was frequently of a much greater degree than that found in simple pneumoconiosis. In a previous work (Thomas, 1951) it was suggested that the LV/RV ratio had to fall below 1, *i.e.*, marked right ventricular hypertrophy had to be present, before electrocardiographic signs of it were seen. This would mean that right ventricular hypertrophy would be detected clinically with greater frequency in massive fibrosis than in simple pneumoconiosis.

In 56 clinical cases which came to necropsy the association of the electrocardiographic signs with the extent of the pneumoconiosis was studied (Table 3). The causes of death were various and not always related to the pneumoconiosis.

TABLE 3

RESULTS OF NECROPSY IN CLINICAL CASES OF PNEUMOCONIOSIS

Stage of Disease	No. of Cases	Marked Right Ventricular Hypertrophy	Pulmonary Heart Disease on E.C.G.
Advanced massive fibrosis ..	32	18	16
Early massive fibrosis ..	17	3	3
Simple pneumoconiosis ..	7	1	1

Thirty-two cases had advanced massive fibrosis (Category C-D, Davies and Mann, 1949). Sixteen of them had electrocardiographic signs of pulmonary heart disease. Marked right ventricular hypertrophy was found at necropsy in all 16 cases. In 14 of these, emphysema of the bullous type and of considerable degree was present, but in the other two with the same degree of right heart hypertrophy emphysema was strikingly absent (Fig. 1). These two cases had the same electrocardiographic signs of right ventricular hypertrophy as similar cases in which emphysema was also present.

A further nine cases with similar advanced massive fibrosis had electrocardiographic changes

which were only suggestive of right cardiac change. Five of these had marked deviation to the right of the electrical axis with prominent pointed P_{II} and P_{III}, two others had the pulmonary P waves only, and two had the pattern of predominant S waves in standard leads I, II, and III. The chest leads did not show the typical signs of right ventricular hypertrophy in these nine cases. Only two hearts of the nine showed marked right ventricular hypertrophy at necropsy. Emphysema was found in six cases. Electrocardiographic change towards the vertical heart pattern with right axis deviation and development of P pulmonale has been present in cases which have shown early right ventricular hypertrophy at necropsy shortly afterwards, and have had severe massive fibrosis. This phase might warrant the use of the term right ventricular stress as a clinical description.

The remaining seven cases of advanced massive pneumoconiosis had a normal QRS pattern and all but one had normal ST segments. In the exception the ST segment was depressed with flat T waves, thought to indicate ischaemic damage, but the muscle was found to be normal and the right ventricle slightly hypertrophied. Adherence of the pericardium may have been responsible for the ST changes in this case.

There were 17 cases with small masses (Category A-B, Davies and Mann, 1949), and it is noteworthy that only three of these produced the electrocardiographic pattern of pulmonary heart disease and these three were the only ones to show right ventricular hypertrophy at the subsequent post-mortem examination. Four of the other cases had some electrocardiographic change ; right axis deviation with pulmonary P waves in three and right bundle branch block in one.

Seven cases of simple pneumoconiosis were studied. In four of them emphysema and bronchitis were the most marked findings and in a fifth there was carcinoma of the lung. One of the four cases with marked emphysema showed right ventricular hypertrophy in the right chest leads and three had pulmonary P waves. One case with simple pneumoconiosis as the principal finding had pulmonary P waves in the electrocardiogram. The last case had auricular fibrillation which in our experience is so uncommon in pulmonary heart disease that another cause was suspected although not found. Of the seven cases, one only showed right ventricular hypertrophy at necropsy, and this had been evident clinically.

The Vascular Lesion

Right ventricular hypertrophy occurs in coalworkers' pneumoconiosis. It occurs with greater frequency and is of greater degree in massive fibrosis than in simple pneumoconiosis. In our material early right ventricular hypertrophy detectable by weighing the ventricles was present in 34% of the massive fibrosis and in 46% of simple pneumoconiosis. In sharp distinction, marked right ventricular hypertrophy, recognizable in the thickness of the right ventricular wall and often detectable during life in the electrocardiogram, was present in 57% of those with massive fibrosis but only in 17% of the cases of simple pneumoconiosis.

In both forms of the disease many cases have other complicating features which may affect the right heart, particularly chronic bronchiolitis and emphysema, but in simple pneumoconiosis even with these marked right ventricular hypertrophy is not frequent. In massive fibrosis cases without these complicating factors produce marked right ventricular hypertrophy. The difference in the incidence of right ventricular hypertrophy suggests that there is a factor operating in massive fibrosis not present in simple pneumoconiosis.

One striking pathological difference between the two disease states is a progressive stenosis of the vessels in the pulmonary arteriolar bed in massive fibrosis (Fig. 2). In and adjacent to the areas of massive fibrosis the pulmonary arterioles showed destruction of the media, invasion of the wall with dust-laden macrophages, and gross intimal thickening with progressive stenosis (Wells, 1954b). The larger the areas of massive fibrosis, the greater the number of vessels involved in the process. This is a slowly progressive stenosing arteriolar lesion and so is capable of producing obstruction to pulmonary blood flow, increased right ventricular work, and eventual right ventricular hypertrophy.

The pulmonary arteriolar stenotic changes are not found in the vessels of the non-fibrosed dust foci of simple pneumoconiosis and right ventricular hypertrophy would not occur from this cause (Fig. 3).

Six cases recorded above with advanced massive pneumoconiosis but without marked right ventricular hypertrophy show a complete absence of the arteriolar lesion in two instances and changes of slight degree in four. This lends support to the association of hypertrophy and the arteriolar lesion although the number is small ; it also helps to explain the occasional anomaly of massive fibrosis present for a long period without signs of right cardiac change. We have observed that there may be a latent period, occasionally up to 20 years, when the lung lesions may be detected and measured radiologically without there being clinical evidence of right cardiac change. The reason for

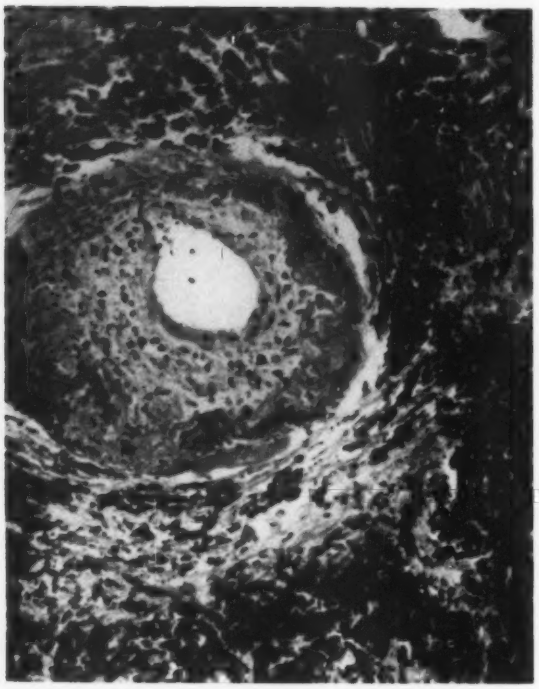

FIG. 2.—The arteriole shown was in a massive dust lesion. The lumen was much reduced by thickening of the vessel. The adventitia and media contained dust-laden macrophages and the structure of the vessel was destroyed. × 155.

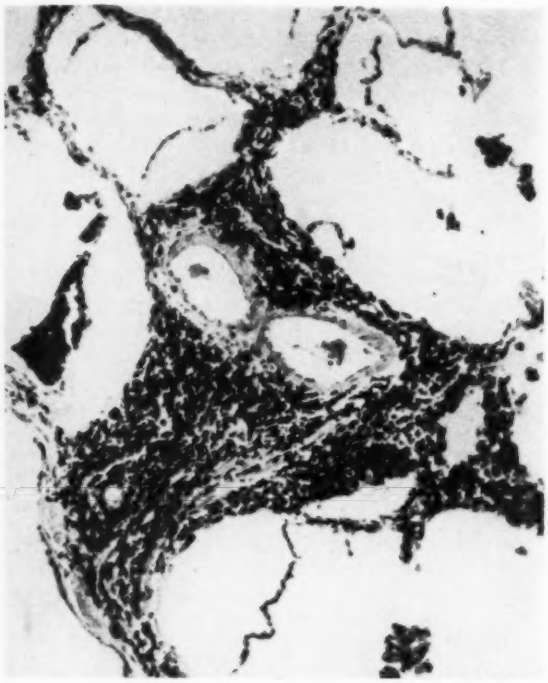

FIG. 3.—The two arterioles shown were in a dust focus from a case of simple pneumoconiosis. The structure of the vessels was normal. × 100.

the absence of pulmonary arteriolar lesions in occasional cases of massive fibrosis is not known. When the pulmonary arteriolar changes have reached a degree sufficient to cause restriction of pulmonary blood flow on exercise and increased right ventricular work then the process becomes irreversible. It is evident that even then years may elapse during which the right ventricle hypertrophies but does not fail. The right ventricular failure comes when there is great hypertrophy and inability to meet the increasing load. In many cases there are the other complicating features—emphysema, bronchiolitis, tuberculosis, or intercurrent disease—which precipitate heart failure earlier or cause anoxic failure or death from other causes. In these cases right heart failure may not be evident and the lesser degree of right ventricular hypertrophy only found if looked for carefully.

The evidence suggests that in simple pneumoconiosis without collagenous fibrosis and without obstructive arteriolar lesions, marked right ventricular hypertrophy is uncommon, and the lesser degrees of hypertrophy which are found are usually in association with chronic bronchiolitis and emphysema.

Electrocardiographic correlation indicates that when right ventricular hypertrophy appears in massive fibrosis it develops to an extent detectable in the electrocardiogram. In simple pneumoconiosis this is not so but many of these cases show a pattern of the vertical heart with tall pointed P waves in leads II and III. It is suggested that this indicates a stage of right heart stress in the presence of pulmonary disease.

Summary

Cardiac hypertrophy in coalworkers' pneumoconiosis has been studied. The overall pattern is presented in an analysis of 1,000 necropsies. The degree of right ventricular hypertrophy has been analysed by weighing the ventricles in 80 cases and comparing its extent with the pneumoconiosis in the lungs. In 56 cases in which clinical details have been available the electrocardiogram and the right ventricular hypertrophy at necropsy are compared.

The evidence suggests that right ventricular hypertrophy is frequent and of considerable degree in massive fibrosis but much less common and of a lesser degree in simple pneumoconiosis. Another difference between the two disease states is in

massive fibrosis a progressive stenosing arteriolar lesion restricting the pulmonary vascular bed, a condition not found in simple pneumoconiosis. This vascular lesion is postulated as the cause of the marked degree of right ventricular hypertrophy in massive fibrosis, and it is shown that this can be so when complicating factors such as emphysema are not present. In simple pneumoconiosis the frequent presence of bronchiolitis and emphysema causes some right cardiac change but not to the same extent.

The electrocardiogram does not identify the early right ventricular hypertrophy although it shows features such as a vertical heart pattern with pulmonary P waves in these cases. In massive fibrosis the electrocardiogram detects the right ventricular hypertrophy when it reaches a marked degree and before heart failure appears.

We wish to thank Dr. J. C. Gilson, Director of the Pneumoconiosis Research Unit (M.R.C.), Llandough Hospital, and members of his staff for permission to study cases under their care and for their help. We are grateful to Professor J. Gough, Department of Pathology, Welsh National School of Medicine, for his advice and criticism.

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RESPIRATORY FUNCTION IN MEN CASTING CADMIUM ALLOYS

PART I: ASSESSMENT OF VENTILATORY FUNCTION

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The ventilatory function of the lungs was assessed in all but four of those workers from the two factories who took part in the investigation described by Bonnell (1955). The maximum ventilatory capacity test and tracings of the expiratory fast vital capacity were used as tests of ventilatory function. The performance of the group of subjects exposed to cadmium fume was compared with that of a control group of subjects who had not been so exposed. Individual performances in these tests, differing widely from the mean performance of the appropriate control group, were then compared with the clinical and radiological findings.

Methods

The tests were performed immediately before or immediately after the clinical examination. Subjects from the control and exposed groups reported in mixed order and, to avoid any possible bias in the conduct of the test, the operator was not informed of the group to which a particular individual belonged.

The following measurements were made on each subject with adequate rest periods between each part of the test.

(1) Vital capacity, five estimations.

(2) Maximum ventilatory capacity (M.V.C.) at 30 and 70 respirations per minute (R.P.M.). These measures are referred to as $M.V.C._{30}$ and $M.V.C._{70}$, the suffix in each case indicating the respiratory rate.

(3) Four expiratory fast vital capacity curves were recorded.

The tests were performed on the spirometer described by Bernstein, D'Silva, and Mendel (1952) which was known to be free from appreciable recording error for the airflows involved.

The vital capacity was taken as the mean of the last four of the five estimates. For the M.V.C. test the subject was asked to breathe in and out of the spirometer as deeply as he was able in time to a metronome. He was asked to breathe through his mouth having been shown how to hold the mask on his face without producing a leak. The operator continually exhorted the subject to breathe more deeply, and helped him by conducting the timing. The recording lasted about 15 seconds. The performance was repeated only if a leak occurred at the face-piece, or if the tidal excursions were grossly uneven, indicating that the subject was not performing to the best of his ability with each breath. It was never necessary to repeat the test at any respiratory rate more than once. All determinations were performed with the subjects standing.

From the mean tidal excursion for each tracing, the M.V.C. and the swept fraction were calculated for each respiratory rate (D'Silva, Freeland, and Kazantzis, 1953; D'Silva and Kazantzis, 1954).

For the expiratory fast vital capacity test the subject was asked to place the mask on his face, inspire as deeply as he was able, and, on receiving a signal, to expire into the spirometer as deeply and as fast as he could. This procedure was repeated four times by each subject. The mean time constant (D'Silva and Kazantzis, 1954) of the four fast vital capacity curves was determined.

Gas volumes were reduced to 37° C. saturated with water vapour at 760 mm. Hg.

Results

Factory A.—Of the 58 men who had worked at some time in the cadmium shop and who were examined clinically, 56 men performed the present series of tests. Sixteen men of this total exposed group had at some time been engaged on casting copper-cadmium alloys (Group 1A, cadmium casting workers). The remaining 40, while working in the same comparatively small shop and being exposed to cadmium fume to a varying degree, had not actually been employed in casting cadmium (Group 1A, vicinity workers). The control group (CA) was made up of 60 men who had never worked in the cadmium shop. They were chosen on an age basis (Bonnell, 1955, Table 1) by the administrative staff of the factory from a considerably larger number of volunteers from other parts of the

TABLE 1

MEAN VALUES AND STANDARD DEVIATIONS FOR AGE, HEIGHT, AND WEIGHT IN THE CONTROL AND EXPOSED GROUPS OF BOTH FACTORIES

Group	Number of Subjects	Age		Height (in.)		Weight (lb.)	
		Mean	S.D.	Mean	S.D.	Mean	S.D.
Factory A							
Control (CA)..	60	49·4	9·30	67·2	2·32	157·6	21·94
Cadmium workers (1A)	16	48·9	8·44	68·0	3·01	154·3	19·74
Vicinity workers (1A)	40	49·0	10·60	67·4	2·33	160·5	27·47
Factory B							
Control (CB)..	44	39·8	12·00	66·4	2·15	139·9	13·97
Cadmium workers (1B)	40	41·4	10·62	66·9	2·99	148·9	25·66

works. All the men in this group performed the present tests. The mean ages, with standard deviations, for the groups described are shown in Table 1. Corresponding figures for the heights and weights are also shown. It can be seen that the groups are similar regarding these three characteristics, and analysis by Student's t test shows no significant differences between the means. Throughout this paper the 5% level of significance is referred to unless otherwise stated but the probability levels are shown in the Tables.

The approximate number of years spent in the casting shop by each individual in the exposed group is shown on a scatter diagram (Fig. 1). It was not possible to assess accurately the number of years spent on the copper-cadmium process by the 16 cadmium casting workers as all of them had also performed other duties in the same shop, but the 16 cadmium workers and 32 of the 40 vicinity workers had been employed in the casting shop for more than 10 years.

The results of the tests for the three groups and for the total exposed group are shown in Table 2. For each, the vital capacity, the $M.V.C._{70}$, the swept fraction at two respiratory rates, and the time constant of the expiratory fast vital capacity curve are shown. The mean value and standard deviation for each group are given. The differences between the mean values for the control and exposed groups were analysed using the t test. For vital capacity, for $M.V.C._{70}$, and for swept fractions, the P values refer to the probability of obtaining a mean value less than the control mean, and for time constant they refer to the probability of obtaining a mean value greater than the control mean. The mean value for the vital capacity and maximum ventilatory capacity is not significantly lower in the total exposed group or its component subgroups compared with the control group. The mean values for the two swept fractions are significantly lower in the total exposed group, and the mean expiratory time constant is significantly higher in the exposed than in the control group. Table 2 shows that when the total exposed group is split into cadmium casting and vicinity workers the values for the swept fractions and time constant differ furthest from the control values in the group of workers casting cadmium. The differences of these means are highly significant. For the vicinity workers the mean value of the swept fraction at 70 R.P.M. is significantly lower, and the mean

FIG. 1.—Approximate number of years spent in the casting shop in the exposed group.

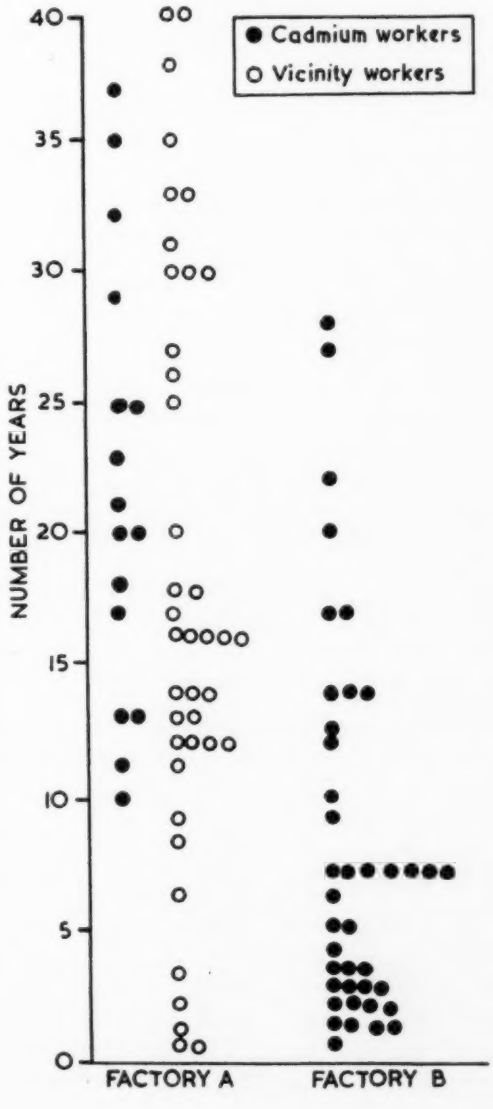

TABLE 2

MEAN VALUES AND STANDARD DEVIATIONS (IN BRACKETS) FOR VITAL CAPACITY, MAXIMUM VENTILATORY CAPACITY AT 70 R.P.M., SWEPT FRACTIONS AT 30 AND 70 R.P.M., AND FOR TIME CONSTANT OF EXPIRATORY FAST VITAL CAPACITY CURVE AT FACTORY A

Test		Control Group (CA)	Total Exposed Group (1A)		Cadmium Casting Workers		"Vicinity" Workers	
Vital capacity		3·99 (0·72)	4·12 (1·06)	0·8>P>0·75	4·46 (1·18)	0·98>P>0·95	3·98 (1·00)	0·5>P>0·4
$M.V.C._{70}$		138 (32)	130 (47)	0·2>P>0·15	132 (51)	0·3>P>0·25	130 (46)	0·2>P>0·1
Swept fraction	at 30 r.p.m.	71·7 (8·83)	67·9 (11·24)	0·025>P>0·01	65·1 (10·30)	P<0·01	69·0 (11·54)	0·1>P>0·05
	at 70 r.p.m.	49·3 (7·75)	44·3 (10·75)	P<0·01	41·1 (8·87)	P<0·001	45·5 (11·15)	0·025>P>0·01
Time constant (sec.)		0·59 (0·16)	0·76 (0·44)	P<0·01	0·84 (0·39)	P<0·001	0·73 (0·46)	0·025>P>0·01

The P values refer to comparisons between each group and the control group.

value for the time constant significantly higher than in the control group.

To summarize the findings in factory A : there is no evidence that exposure to cadmium affects the vital capacity adversely, and the groups cannot be distinguished by means of their maximum ventilatory capacities. The swept fractions at the two respiratory rates are significantly lower in the exposed group, and the time constant of the expiratory fast vital capacity curve significantly greater. These measures thus provide a method for differentiating between the means of the control and exposed groups.

Factory B.—The exposed group (1B) was made up of 40 of the 42 men who took part in the clinical examination. Thirty-six of these men were also examined by Buxton (Part II). The control group (CB), composed of subjects performing the same kind of work casting metals but who had never been exposed to cadmium fume, was made up from workers volunteering for examination from the brass and iron foundries. As in Factory A, they were picked by the administrative staff on an age basis (Bonnell, 1955, Table 1). All 44 men who were clinically examined performed the lung function tests, and, of these, 37 men were examined by Buxton. The means and standard deviations for age, height, and weight, are given in Table 1. The groups are homogeneous in these respects, and no significant differences can be detected with the t test.

The number of years spent by each individual in the casting shop is shown in the scatter diagram (Fig. 1). The pattern of exposure is different from that in Factory A. In Factory B 28 men out of 40 worked in the cadmium casting shop for less than 10 years, 12 men had been exposed for from 10 to 28 years. Also, the type of exposure in the two factories was not comparable, as the casting process in Factory B was different, and the workshop was much larger than that in Factory A.

TABLE 3

MEAN VALUES AND STANDARD DEVIATIONS (IN BRACKETS) FOR VITAL CAPACITY, $M.V.C._{70}$, SWEPT FRACTIONS AT 30 AND 70 R.P.M., AND FOR TIME CONSTANT OF EXPIRATORY FAST VITAL CAPACITY CURVE AT FACTORY B

Test		Control Group (CB)	Total Exposed Group (1B)		>10 Years' Exposure		<10 Years' Exposure	
Vital capacity		4·46 (0·69)	4·32 (0·82)	0·25>P>0·2	3·95 (1·03)	0·02>P>0·01	4·47 (0·68)	P>0·05
$M.V.C._{70}$		148 (29)	142 (50)	0·3>P>0·25	118 (50)	P<0·01	153 (47)	P>0·05
Swept fraction	at 30 r.p.m.	70·0 (9·19)	67·3 (14·03)	0·25>P>0·2	61·6 (14·96)	P<0·01	69·7 (13·14)	P>0·05
	at 70 r.p.m.	47·5 (8·35)	46·2 (12·29)	0·3>P>0·25	41·8 (12·58)	0·05>P>0·02	48·1 (11·87)	P>0·05
Time constant (sec.)		0·58 (0·26)	0·79 (0·63)	0·02>P>0·01	0·98 (0·82)	P<0·01	0·71 (0·52)	P>0·05
Age		39·8 (12·00)	41·4 (10·62)	P>0·05	49·8 (9·11)	P<0·01	37·8 (9·20)	P>0·05

The P values refer to comparisons between each group and the control group.

The results for the two groups are shown in Table 3. For each group, the mean and standard deviation are given for vital capacity, for $M.V.C._{70}$, for the swept fraction at the two respiratory rates, and for the time constant of the expiratory fast vital capacity curve. For each measurement, the significance of the difference between the means in the exposed and control groups was tested by the t test. No significant differences between the means were found for vital capacity, maximum ventilatory capacity, or swept fraction, but the mean time constant of the expiratory fast vital capacity curve was significantly longer in the exposed group. The trend of the means in the other observations is, however, in the expected direction and the scatter around the mean is greater in the exposed group.

When the exposed group is divided into those subjects with 10 years' and over and less than 10 years' exposure, significant differences are found between the control group and those exposed over 10 years (Table 3). However, the subjects with the longer exposure were the older members of the group so that the mean age of the 12 subjects comprising the maximally exposed group was about 10 years greater than that of the control group or of the group exposed for less than 10 years. The effect of age on performance in the tests had therefore to be ascertained before differences could be attributed to exposure to cadmium fume.

Partial regression coefficients on age and on years of exposure to cadmium were calculated from the pooled data of the two groups, treating the control group as having zero exposure (Table 4). From the partial regression coefficients it was concluded that between the control and maximally exposed groups, differences in the means for vital capacity, $M.V.C._{70}$, and swept fraction at 30 and 70 R.P.M. could be explained by the effect of age. For the time constant of the expiratory fast vital capacity curve, a significant regression on exposure was demonstrated, the regression on age not reaching the 10% level of significance. For the time constant, the difference between the groups is therefore likely to be due, at least in part, to exposure to cadmium as distinct from age.

Individual Performances in the Two Factories Compared with Clinical and Radiological Findings

Individual performances have been compared with the mean performance of the control group in the factory. Values for the swept fraction lower than twice the standard deviation from the mean, or for the time constant greater than twice the standard deviation from the mean for the control group, have been selected for comparison with clinical and radiological findings. The limits set are different in the two factories owing to the different values obtained for the control groups in each, but the differences are small. Values outside these limits in the two factories are shown in Tables 5 and 6, a blank space denoting a value within twice the standard deviation from the mean. The diagnosis given in the Table was made at the end of the clinical examination, and is not necessarily identical with the final diagnosis given by Bonnell, which was arrived at after consideration of clinical, respiratory function, and radiological findings (Bonnell, 1955).

Factory A.—Table 5 shows subjects who have swept fractions less than 54% at 30 R.P.M., or 34% at 70 R.P.M., or who have an expiratory fast vital capacity curve with a time constant greater than 0·91 second. The first six of these subjects (A5, A8, A10, A12, A13, A16) had values outside the limits described on all three counts. All gave histories of increasing dyspnoea on exertion for a number of years. Radiological appearances in four out of the six were suggestive of emphysema (A10 was not radiographed). Two subjects (A14, A15) had one swept fraction and a time constant outside

TABLE 4

SIGNIFICANCE OF PARTIAL REGRESSION COEFFICIENTS ON AGE (b_1) AND EXPOSURE TO CADMIUM FUME (b_2) IN FACTORY B

			Regression Coefficient		Standard Error	t	P (One-sided Test)
Vital capacity		b_1	−0·039	±	0·0063	6·23	P<0·001
		b_2	−0·0068	±	0·011	0·60	0·3>P>0·25
$M.V.C._{70}$		b_1	−1·77	±	0·35	5·06	P<0·001
		b_2	−0·80	±	0·63	1·27	0·15>P>0·1
Swept fraction	at 30 r.p.m.	b_1	−0·36	±	0·11	3·21	P<0·01
		b_2	−0·26	±	0·20	1·30	0·1>P>0·05
	at 70 r.p.m.	b_1	−0·17	±	0·11	1·59	0·1>P>0·05
		b_2	−0·25	±	0·19	1·31	0·1>P>0·05
Time constant		b_1	+0·0056	±	0·0048	1·17	0·15>P>0·1
		b_2	+0·019	±	0·0085	2·20	0·025>P>0·01

TABLE 5

SWEPT FRACTIONS AND TIME CONSTANTS FALLING OUTSIDE TWICE THE STANDARD DEVIATION FROM THE MEAN FOR CONTROL GROUP WITH RADIOLOGICAL AND CLINICAL FINDINGS AT FACTORY A

Case No.	Age	Years' Exposure	Swept Fraction		Time Constant (sec.)	Radiological Findings	Clinical Notes on Respiratory System	Clinical Diagnosis
			At 30 R.P.M.	At 70 R.P.M.				
A5	58	18	46·2	25·6	2·08	Some signs of emphysema	2 yr. dyspnoea, chronic winter cough	Emphysema
A8	55	20	47·9	31·0	1·21	Emphysema	4 yr. dyspnoea	Emphysema
A10	42	17	51·2	30·8	1·11	Not radiographed	3 yr. dyspnoea, cough	Emphysema
A12	54	10	37·6	27·3	3·13	Emphysema	10 yr. dyspnoea, 3 yr. winter cough	Emphysema
A13	61	8	49·8	25·2	1·47	Emphysema	5 yr. dyspnoea, 8–10 yr. chronic winter cough	Emphysema
A14	64	13	—	28·6	0·93	Nil	2 yr. dyspnoea, many yr. cough	Chronic bronchitis
A15	45	18	—	29·0	1·28	Nil	Many yr. chronic bronchitis and asthma	Nil
A16	50	33	50·8	27·8	1·13	Nil	9 yr. dyspnoea, ? functional element	Nil
A17	60	13	—	—	0·94	Nil	Nil	Nil
A18	64	13	—	33·8	—	Nil	Nil	Nil
A20	40	20	—	30·8	—	Nil	2 yr. chronic winter cough	Nil
A21	64	Control	—	—	1·12	Pneumoconiosis	Chronic winter bronchitis	Nil
A22	54	Control	48·1	34·0	—	Some signs of emphysema	Nil	Nil
A23	69	Control	48·8	—	—	Localized emphysema R. upper zone only	Nil	Nil
A1	56	30	—	—	—	Emphysema	Unwell 6 mth., progressive dyspnoea	Emphysema
A2	52	13	42·3	24·4	2·39	Emphysema	7 yr. progressive dyspnoea, cough 4 yr.	Emphysema
A3	58	11	29·5	16·7	2·45	Emphysema	10 yr. dyspnoea	Emphysema
A4	49	12	29·8	16·8	4·00	Emphysema	6 yr. dyspnoea, cough, and sputum	Emphysema

the limits set. In six subjects (A17, A18, A20, A21, A22, A23) either the time constant or one or more swept fractions fell outside the adopted limits, but not both time constant and swept fractions. Three of these six subjects belonged to the control group. No member of the control group appeared outside the limits on all counts.

Radiological abnormalities were diagnosed in nine subjects (not shown in Table 5) whose performance in the tests fell within the limits described above. Seven of these subjects, three from the control and four from the exposed group, were considered by the radiologist to show some signs of emphysema. The remaining two showed evidence of pneumoconiosis. In none of the seven cases was an unqualified diagnosis of emphysema made, the radiographs being commented on as showing "some signs of emphysema", "slight emphysema", or "localized emphysema". On clinical examination one of these nine subjects was diagnosed as having emphysema, the other eight were passed as normal. Two subjects from the exposed group diagnosed clinically as having emphysema and four from the control group diagnosed with chronic bronchitis showed no abnormality on radiological examination and performed within the described limits in the ventilatory function tests.

Four men who had worked on the copper-cadmium process for a number of years but who were on sick leave during the survey were not included in the exposed group. They were investigated at the London Hospital during the succeeding six months, the findings being reported in full by Bonnell (1955). Clinical and radiological diagnoses of emphysema were made in all four cases. The results in the ventilatory function tests were outside the limits described for three out of the four subjects on all counts. The findings in these four men (A1, A2, A3, A4) are included in Table 5.

Factory B.—Subjects with a swept fraction of less than 52% at 30 R.P.M. or 31% at 70 R.P.M., and those with an expiratory fast vital capacity curve with a time constant greater than 1·10 seconds are shown in Table 6 together with clinical and radiological findings. Four subjects from the exposed group (B1, B3, B4, B6) gave values outside these limits for both swept fractions and for the time constant. Subsequent comparison with the clinical findings showed that all four gave histories of progressive dyspnoea on exertion and in two of these the radiological findings were suggestive of emphysema. The one control subject (B12), all of whose values fell outside the above limits, was passed as normal on clinical and radiological examination. One subject from the exposed group (B2) had a time constant and one swept fraction

TABLE 6

SUBJECTS WITH SWEPT FRACTION OR TIME CONSTANT OUTSIDE THE DESCRIBED LIMITS COMPARED WITH CLINICAL AND RADIOLOGICAL FINDINGS AT FACTORY B

Case No.	Age	Years' Exposure	Swept Fraction		Time Constant (sec.)	Radiological Findings	Clinical Notes on Respiratory System	Clinical Diagnosis
			At 30 R.P.M.	At 70 R.P.M.				
B1	52	10	30·6	17·1	3·27	Nil	15 yr. dyspnoea, chronic cough	Emphysema
B2	49	27	50·8	—	1·33	Emphysema	Nil	Nil
B3	43	9	36·0	17·1	3·02	Nil	5 yr. dyspnoea, chronic cough	Emphysema
B4	58	22	44·0	27·2	1·38	Localized emphysema	3 yr. dyspnoea, chronic cough	Emphysema
B5	59	17	—	—	1·50	Emphysema probable	6 mth. dyspnoea (increasing)	Emphysema
B6	50	7	30·6	16·3	1·50	Emphysema	1 yr. dyspnoea, cough, sputum	Emphysema
B11	39	14	—	28·5	—	Nil	Nil	Nil
B12	39	Control	47·9	30·1	1·88	Nil	20 yr. ago pleurisy now N.A.D.	Nil
B13	54	Control	50·6	—	—	Nil	Many years cough and sputum	Nil
B14	53	Control	—	—	1·12	Emphysema	Nil	Nil
B15	58	Control	—	25·1	—	Nil	Felt giddy during tests	Nil

outside the limits, and five subjects (B5, B11, from the exposed; B13, B14, B15, from the control group) had either time constant or one swept fraction outside these limits but not both.

Abnormalities were diagnosed on radiological grounds in two subjects whose performance in the ventilatory function tests fell within the limits described. One subject was passed as normal on clinical examination, the other gave a history of asthma.

In summary, in Factory A nine subjects who had been exposed to cadmium gave values outside the limits set on the three counts considered. A clinical diagnosis of emphysema was made in eight of the nine cases, and a radiological diagnosis of emphysema in seven of the nine cases.

In Factory B four subjects from the exposed and one from the control group gave values outside the limits set on the three counts considered. A clinical diagnosis of emphysema was made in the four from the exposed group and a radiological diagnosis of emphysema in two of those four subjects.

Comment

The subjects comprising the control groups in this investigation were not selected on the basis of freedom from disease, so that a similar incidence of respiratory disorder may be expected to occur in the control groups as in the populations from which these groups were drawn. The significant differences between the results of the tests of ventilatory function obtained from the control and exposed groups suggest that these groups differ with respect to some factor affecting the incidence of disease. The only factor that we know to be different in the two groups is the environmental one, namely, exposure to cadmium fume.

In certain forms of ventilatory impairment, as in asthma and emphysema, the M.V.C. is reduced disproportionately to any reduction in vital capacity (Gaensler, 1951), so it is in distinguishing this form of ventilatory impairment that the swept fraction would be of most use. A decrease both in the vital capacity and in the M.V.C. produced by a restriction of rib movements in normal subjects has been shown not to reduce the swept fraction (D'Silva and others, 1953).

The tidal excursion in the M.V.C. test at rates between 20 and 100 R.P.M. has been shown (Bernstein and Kazantzis, 1954) to be made up of portions of the inspiratory and expiratory fast vital capacity curves. It is reasonable to assume that a slowing of either inspiration or expiration, or of both processes, would lead to a reduction in swept fraction. From Tables 5 and 6 it can be seen that the majority of subjects with low swept fractions also had long time constants for their expiratory fast vital capacity curves. It may be that in some instances a low swept fraction may be due to the association of a normal expiratory curve and a slow inspiratory curve, while in other instances a slow expiratory curve may be compensated by a fast inspiratory curve, thus giving a normal swept fraction. Expiratory fast vital capacity curves were obtained from all subjects, but the importance of the inspiratory curve was not recognized at the time of this investigation.

The selection of abnormal individual performances presents difficulties when there is much individual variation within the limits of normal, and when, as happens with most tests, normal and abnormal populations overlap to a greater or lesser extent. The difficulties are greater when the test

requires the cooperation of the subject, as in this instance.

In this investigation subjects with one or more low swept fractions, or a long time constant compared with the performance of the control group, have been selected for comparison with the clinical and radiological findings. The division between an "abnormal" and a "normal" performance was set at twice the standard deviation from the mean performance for the control group, so that about one observation in 40 in a sample from the control group population would be expected, without allowing for individual age differences, to fall below the lower limit for the swept fraction, or above the upper limit for the time constant.

A single low swept fraction or a long time constant could also be due to an extraneous influence during the test, such as a diversion of the subject's attention, inhalation of saliva, or a desire to cough. Two low swept fractions and in addition a long time constant in the same subject are unlikely to be due to such a cause. Of the time constants recorded for 21 subjects in Tables 5 and 6, 17 were greater than the mean plus three times the standard deviation. Only about one subject in 200 from the control group would be expected to fall outside these limits.

Tables 5 and 6 show that the subjects who performed "abnormally" on all the tests showed clinical evidence of emphysema. Not all the subjects with low results in one or two tests showed clinical evidence of respiratory disease, and there were three subjects diagnosed clinically as having emphysema who performed within the limits described on all the tests.

Difficulties involved in the purely clinical diagnosis of emphysema have been discussed by Fletcher (1952) and in the radiological diagnosis of emphysema by Knott and Christie (1951). The assessment of the ventilatory function of the lung taken in conjunction with other findings may be of considerable value in arriving at a diagnosis. It is believed that the methods described in this paper provide a more sensitive index of ventilatory impairment than those hitherto used.

Summary

Vital capacity, maximum ventilatory capacity at 30 and 70 R.P.M., and expiratory fast vital capacity records have been obtained from groups of workers exposed to cadmium fume in two factories and from control groups with similar age distributions. From this data, swept fractions (at the respiratory rates investigated) and time constants of the expiratory fast vital capacity curves have been obtained.

Measurements of the swept fractions and of the time constants of the expiratory fast vital capacity curves have proved useful in differentiating between the control and the exposed groups.

Standards of normality based on the performance of the control group have been laid down, and individual performances outside the 5% confidence limits have been compared with the clinical and radiological findings.

The significance of the swept fraction and the time constant, and the measure of agreement with the clinical and radiological findings have been discussed.

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PART II: THE ESTIMATION OF THE TOTAL LUNG VOLUME, ITS SUBDIVISIONS, AND THE MIXING COEFFICIENT

BY

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Method

The apparatus used, which in principle employed a closed circuit helium technique with a katharometer, was a modification of that described by Bates and Christie (1950). The side circuit carbon dioxide absorber was omitted, and crushed ice was placed in the spirometer jacket 20 minutes before the start of the experiment. This reduced the rise in temperature in the spirometer. The subject, who was seated in an upright chair, was instructed to breathe normally. He inhaled pure oxygen from a Douglas bag for seven minutes and expired into

the atmosphere so that the nitrogen of his functional residual air was replaced by oxygen. At the end of a normal expiration he was connected to the spirometer circuit of 4 litres capacity and containing about 10% helium in oxygen. The gas was circulating at the rate of 50 to 60 litres a minute but the flow through the katharometer was only 0·75 litre per minute.

After a latent period of five to 15 seconds the galvanometer needle started to move, the time lag depending on the katharometer response and the respiratory rate of the subject. The movement was followed by a tracking device as described by Bates and Christie (1950). When the needle ceased to move, showing that mixing was complete, the helium concentration was read. At least two vital capacities were then recorded and the volumes calculated for 37° C. and saturated with water vapour.

From the above data, the total lung volume and its subdivisions were calculated. The mixing efficiency was calculated and scaled up by 100/80 in the manner described by Bates and Christie (1950).

Experimental Subjects

Those workers directly exposed to cadmium fumes were studied together with a control group drawn from the brass and iron foundries in the same factory, which was called factory B by Bonnell (1955). The selection of the control group has been discussed by Bonnell (1955). At the time of the clinical examination appointments were made for these respiratory function tests so that more often than not they took place on a different day from the clinical examination. The observer did not know at the time of the test from which group the subject was drawn. One test was made on each individual. In the "exposed" group 37 men out of 42 were studied, five failing to attend, and 36 out of 44 in the control series, eight defaulting.

The ages of the exposed and control groups ranged from 23 to 65 (mean 41·9 years) and 18 to 65 (mean 40·9 years) respectively. Of the men exposed to cadmium fumes, 13 (average age 49·5 years) had been exposed for more than 10 years (cadmium group with a long exposure), and 24 (average age 37·8 years) for less than 10 years (cadmium group with a short exposure).

Results

Subdivisions of the Total Lung Volume.—Table 1 shows the results obtained and also the probability that the observed differences between the various pairs of means would be exceeded by chance. It will be seen that the mean total lung volume in the three groups (columns 2, 3, and 4) was not significantly different. The mean vital capacity of the subjects in the cadmium group with a long exposure (column 3) was significantly less than of those in the control group (column 2) while the value for the cadmium group with a short exposure (column 4) differed little from the control group. The mean functional residual air was similar in the three groups (columns 2, 3, and 4), but the mean residual air, and the residual air expressed as a percentage of the total lung volume, were significantly greater in the group of men exposed to cadmium for more than 10 years than in the control series (columns 3 and 2).

It has been noted above that there was a considerable difference between the mean ages for the control group and the cadmium group with a long exposure. It was, therefore, necessary to study the relationship between age and the various subdivisions of the lung volume before ascribing the significant differences to other factors.

Total Lung Volume and Age.—There was no regression of significance ($P > 0·7$) of total lung volume on age so that the values recorded in Table 1 for the total lung volume are directly comparable.

TABLE 1

MEAN VALUES FOR SUBDIVISIONS OF TOTAL LUNG VOLUME OF WORKERS EXPOSED TO CADMIUM FUME AND CONTROL SUBJECTS

1	2	3	4	5	6
				Values of P for	
	Controls	Exposed >10 Years	Exposed <10 Years	Controls and Cadmium Group with Long Exposure	Controls and Cadmium Group with Short Exposure
Number of Cases	36	13	24	—	—
Age mean	40·9	49·5	37·8	—	—
S.D.	12·3	8·3	8·4	—	—
Total lung volume	6·76	6·81	6·98	>0·4	>0·15
S.D.	0·84	0·89	1·1	—	—
Vital capacity	4·38	3·79	4·39	<0·005	>0·45
S.D.	0·58	0·86	0·74	—	—
Functional residual air	3·71	3·80	3·76	>0·35	>0·4
S.D.	0·85	0·93	1·1	—	—
Residual air	2·38	2·99	2·59	<0·01	>0·15
S.D.	0·72	0·78	0·99	—	—
Residual Air × 100 / Total Lung Volume	34·57	43·94	36·59	<0·005	>0·2
S.D.	8·3	10·3	10·9	—	—

Vital Capacity and Age.—In the control group the vital capacity tends to diminish with increasing age, and the relationship is significant ($P < 0.01$). If parallel lines are fitted to the control group and the cadmium group with long exposure, the difference in position of the two lines does not quite reach the 5% level ($P = 0.06$ on a one-sided test). Here and at corresponding points in the analysis of the other tests, a one-sided test of significance is employed since exposure to cadmium could hardly be expected to improve respiratory function. If, however, regression lines are fitted separately to the two groups, they are found to differ significantly in their slope ($P = 0.01$), the exposed group having the larger negative regression coefficient. This is due mainly to a number of men at the upper end of the age range who had especially low vital capacities. It may therefore be concluded that for the higher ages at least some factor other than age was responsible for the decrease in vital capacity, and, as the results from the cadmium group with a short exposure did not demonstrate such a difference, the greater length of exposure to cadmium may well be a factor in its production.

Functional Residual Air and Age.—Since there was no significant regression ($P > 0.1$) in the values of functional residual air and age, their further manipulation in Table 1 is not required. The results for the cadmium group with long and short exposure and the control group remain not significantly different.

Residual Air and Age.—The volume of the residual air tends to increase with increased age, the slope of the line for the control group being significant ($0.01 < P < 0.05$). Lines fitted to the control group and to the cadmium group with a long exposure did not differ significantly in slope ($0.1 < P < 0.2$). The difference in position, however, of the parallel lines is on the border of significance ($P = 0.05$).

Residual Air as a Percentage of Total Lung Volume and Age.—The residual air expressed as a percentage of total lung volume tends to increase with age. The difference in slope of lines fitted separately to the control group and the cadmium group with a long exposure does not quite reach the $P = 0.05$ level of significance. The slope of the lines is, however, highly significant ($P < 0.01$), and their positions differ significantly ($P = 0.01$), the mean values in the exposed group being higher at any given age. This difference is not shown by the cadmium group having a short exposure so that the length of exposure to cadmium may be a causative factor.

Mixing Efficiency.—The mixing coefficients were calculated by the method of Bates and Christie (1950). The mean for the control group (49·3%, S.D. 10·0) and that for the cadmium group with a long exposure (53·1%, S.D. 15·8) showed a difference which was barely significant ($P > 0.05$). The cadmium group with a short exposure had a mean of 49·5% (S.D. 16·7) which did not differ significantly ($P > 0.4$) from the control figure. There was no significant regression of the mixing coefficient on age ($P > 0.5$) in the control group.

Assessment of Individual Workers.—Consideration of individuals in a group is statistically less easy than making an appraisal of the group as a whole. The control group in this investigation was selected in a manner which was considered to allow a fairer assessment of the cadmium workers in relation to the general population of similar age than if any arbitrary standard of fitness had been employed. After making allowance for age, the limit of normality has been taken as the sum or difference of twice the standard deviation and the mean of the control group, or, where applicable, twice the standard deviation from regression. This means that one in 40 of the control observations can be expected to fall outside the limits set. Table 2 shows only those subjects in the control series and in the groups exposed to cadmium whose results for the total lung volume, its subdivisions, the relative percentage of residual air to total lung volume, or for the mixing coefficient, fall outside the limit.

It will be observed that of 36 subjects in the control group, 33 were within the limits set in every test. Nos. B14 and B34 were each outside this range in one test only, an observation which can well be ascribed to random error. Subject B30, aged 25 years, had a past history of a spontaneous pneumothorax without other evidence of chest disease, an occurrence which could account for the abnormal figures obtained in tests 2 and 4.

Of the 13 individuals in the cadmium group with a long exposure, nine gave results within the limits adopted in all six tests. No. B5's performance in test 4 may well have been influenced by random errors. But three out of 13 had low figures in test 2, and three out of 13 high values in test 5 which is significant as each of the subjects B31, B4, and B1 gave abnormal results in both tests.

In the cadmium group exposed for a shorter period, 18 of 24 subjects gave results within the range adopted as normal in each of the six tests. Subject B38 suffered from bronchial asthma of some years' duration and abnormal findings would be expected, so that he need be considered no further

TABLE 2
RESULTS ADJUSTED FOR AGE IN CASES WITH ABNORMAL FINDINGS

Group	Case Number	1 Total Lung Volume	2 Vital Capacity	3 Functional Residual Air	4 Residual Air	5 Residual Air × 100 / Total Lung Volume	6 Mixing Coefficient
Control	B30	—	Low	—	High	—	—
	B14	—	—	High	—	—	—
	B34	—	—	—	—	—	Low
Cadmium with long exposure	B31	—	Low	—	—	High	—
	B4	—	Low	—	—	High	—
	B5	—	—	—	High	—	—
	B1	—	Low	—	—	High	—
Cadmium with short exposure	B6	—	Low	—	High	High	—
	B33	High	—	High	High	High	Low
	B35	—	Low	—	—	—	—
	B36	High	—	—	High	—	—
	B37	—	Low	—	—	—	—
	B38	High	—	—	High	High	—

" High " signifies a value greater, and " low " less, than the limit set, namely, twice the standard deviation from the control mean, or, where applicable, twice the standard deviation from regression.

in this paper. Nos. B35 and B37 had low vital capacity figures which can be ascribed to chance factors operating. Subject B36 performed poorly in tests 1 and 4, No. B6 in tests 2, 4, and 5, while No. B33 had a normal figure for his vital capacity only.

The tests listed in Table 2 are not completely independent of one another in a statistical sense, and the degree of their interdependence cannot be assessed. It would therefore be difficult to calculate the probability of a given subject producing abnormal results in more than one test by operation of the laws of chance.

Comment

In the present investigation no single subject had abnormal figures for the total lung volume and all four of its subdivisions when compared with the control group. The residual air expressed as a percentage of the total lung volume was high in some cases (Table 2). This has been suggested by Hurtado and Boller (1933), Friberg (1950), and Bates and Christie (1950) as a reliable aid in the diagnosis of emphysema. As yet there is no precise information as to the factors on which the volume of the residual air, the functional residual air, the vital capacity, or the total lung volume depend. In any series of normal subjects the size of this fraction, of the volume of the residual air, and of the total lung volume vary widely so that there is no theoretical reason for supporting the assumption that a value for this fraction of over 42% (Bates and Christie, 1950) or over 35% (Hurtado and Boller, 1933) is evidence of emphysema. Until the factors on which the total lung volume and its subdivisions depend are known, the only sound method of assessing results is on a statistical basis. This applies particularly to this study since the nature of the alteration in function of the lungs which may occur with exposure to cadmium fume has not been fully worked out.

In comparing these results with those of Kazantzis in Part I it should be noted that the control group in this paper contained 36 men all of whom featured in his series. But in the group exposed to cadmium the five defaulters all belonged to the group exposed for less than 10 years, while Kazantzis had one defaulter in each exposed group. The difference in vital capacity figures in the two papers could not be explained by the difference in the number of subjects involved, although Kazantzis' deductions are drawn from the results of the exposed group as a whole, while the conclusions in this paper are based on the figures obtained from the 13 men exposed to cadmium for more than 10 years. Further, his subjects were examined in the standing position, and, in this study, sitting in a chair. It may be therefore that posture played a part in producing the discrepancies, and different characteristics of the apparatus may also be involved. This would only be the case if any disability were accentuated by these factors.

The measure of agreement between the clinical assessment and the performance in the various tests of respiratory function may be seen in Table 3. Kazantzis' three tests are the swept fractions at 30 and 70 respirations per minute and the time constant for the expiratory fast vital capacity curve. The author's six points are the total lung volume, the vital capacity, the functional residual air, the residual air, the residual air expressed as a percentage of total lung volume, and the mixing coefficient. In neither series of tests are the variables independent of one another. Of the workers exposed to cadmium

TABLE 3
CORRELATION OF ABNORMAL RESULTS

Group	Case Number	Clinical Findings	Radiological Findings	Results of Respiratory Function Tests	
				Kazantzis	Buxton
Control	B30	—	—	—	2 6
	B14	—	E	1 3	1 6
	B34	—	—	—	1 6
	B13	—	—	1 3	—
	B32	—	E	—	—
	B12	—	—	3 3	—
	B15	—	—	1 3	—
	B39	—	E	—	—
Cadmium with long exposure	B31	—	—	—	2 6
	B4	E	E	3 3	2 6
	B5	E	E	1 3	1 6
	B1	E	—	3 3	2 6
	B11	—	—	1 3	—
	B2	—	E	2 3	—
Cadmium with short exposure	B6	E	E	3 3	3 6
	B35	—	—	—	1 6
	B33	—	—	—	5 6
	B36	—	—	—	2 6
	B37	—	—	—	1 6
	B38	—	E	—	3 6
	B3	E	—	3 3	—

E = emphysema. Kazantzis' results are obtained from page 32.

fume in factory B, five were diagnosed clinically as having emphysema. Four of these (B1, B4, B3, and B6) were found to be abnormal in each of Kazantzis' three tests, and three had an increased residual air : total lung volume ratio, while subject B5 had a residual air figure outside the limit set. The other abnormal figures for the residual air expressed as a percentage of the total lung volume were obtained from subjects B31 and B33 who were clinically normal and produced normal values in the tests of ventilatory function. The effect of choosing a population which was unselected on the grounds of health may affect the standards adopted in the ventilatory tests and the gas distribution procedures differently. Further it would be expected that where the pathological condition existed in a less severe degree this might be detected in one set of tests but not necessarily in all the tests. In consequence it is not unexpected that the results of the respiratory function tests correlated satisfactorily in the severely affected cases with the clinical and radiological assessment but poorly where the condition was less marked.

Summary

This survey was composed of 36 control subjects and 37 men who had been exposed to cadmium fume.

Estimation of the total lung volume, its subdivisions, and of the mixing coefficient was made in each case.

The 13 workers whose exposure to cadmium was of more than 10 years' duration performed less well when compared as a group than the control series.

The difference between the 24 workers whose exposure to cadmium was less than 10 years and the control group was not significant.

In each of the two exposed groups three subjects produced results which were abnormal and unlikely to be chance findings.

It was concluded that these tests of respiratory function showed that the group exposed to cadmium for more than 10 years differed significantly from the control group, that certain individual workers gave abnormal figures and that these differences could not be ascribed to chance.

It gives me great pleasure to acknowledge the help of Professor J. L. D'Silva. My thanks are also due to Dr. P. Armitage for statistical advice and to Miss Audrey Murden for technical assistance.

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THE ACTION OF VARIABLE AMOUNTS OF TRIDYMITE, AND OF TRIDYMITE COMBINED WITH COAL, ON THE LUNGS OF RATS

BY

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It was first shown by Gardner (1938a and b) in animal experiments that tridymite appeared to be more fibrogenic than quartz when tested by intravenous injection in rabbits. In fact, all his animals injected with tridymite died by 11 weeks. King, Mohanty, Harrison, and Nagelschmidt (1953a) used intratracheal injections into the lungs of rats, and compared well graded pure samples of quartz and tridymite, with the result that tridymite produced grade 5 fibrosis after nine weeks as against 38 weeks for quartz. The tridymite sample used had been in contact with hydrofluoric acid during purification, and as it had been found in other experiments (King and others, 1953b) that quartz etched with hydrofluoric acid also was more fibrogenic than normal quartz, there remained a suspicion that a trace of hydrofluoric acid had been the cause of the extra activity of tridymite.

A new series of experiments was therefore set up with another sample of tridymite which had never been in contact with hydrofluoric acid. This sample was used at three different levels of dosage (12·5, 25, and 50 mg. per rat), and electro-dialyzed tridymite which was free from any adsorbed ions was also used at the 25 mg. per rat level.

The fibrogenic activity of quartz and other materials has in the past been expressed solely on the basis of the degree of maturity of the lesions and the following five grades of fibrosis were used : grade 1, loose reticulin fibres with no collagen formation ; grade 2, compact reticulin fibres with or without collagen formation ; grade 3, slightly cellular but mostly collagenous ; grade 4, wholly collagenous and completely acellular ; and grade 5, acellular, collagenous and confluent.

It has been felt for some time that this was not entirely satisfactory for quantitative assessments because the amount as well as the degree of fibrosis should be measured. Various attempts at an area assessment from microscope slides, and to use radiography of air-inflated rats' lungs, did not prove successful, but a quantitative estimation of collagen (Stacy and King, 1954) from dried lung material proved to be possible, and this technique was used to measure the fibrogenic activity of tridymite. Similar data for quartz were available, and this allowed equal dosages of quartz and tridymite to be compared.

It had been found previously (Ray, King, and Harrison, 1951) that 2 mg. of quartz did not produce any fibrosis in the lungs of rats, but did produce fibrosis of the lymph nodes, into which all of it appeared to have been moved, when given alone. A mixture of 2 mg. of quartz and 98 mg. of coal did, however, produce fibrosis up to grade 2 after five months in the lungs of rats. Similar experiments with smaller amounts of dust were also made with tridymite and two samples of coal.

DESCRIPTION OF SAMPLES

Tridymite

This was prepared from Lochaline sand by the use of a flux. The flux was dissolved after heating by prolonged boiling with hydrochloric acid. The resulting tridymite was very pure, and a fraction below 2 μ was prepared by sedimentation in water after grinding the material in an agate mortar. Part of the tridymite was electro-dialyzed for 24 hours between platinum electrodes to remove any adsorbed electrolyte. The chemical analysis, solubilities, and particle size distribution of the two samples of tridymite are given in Table I.

TABLE 1
PARTICLE SIZE DISTRIBUTION AND ANALYSIS OF SAMPLES OF TRIDYMITE

Sample	Tridymite		Electro-dialyzed Tridymite	
Size in μ	Percentage		Percentage	
	By No.	By Mass	By No.	By Mass
0·23–0·32	—	—	22·4	0·3
0·32–0·45	12·5	0·7	22·1	0·9
0·45–0·64	22·4	3·4	18·4	2·1
0·64–0·9	29·4	12·3	14·1	4·4
0·9–1·3	27·3	31·8	9·4	8·3
1·3–1·8	6·2	20·7	7·8	19·6
1·8–2·6	1·6	15·0	4·0	28·2
2·6–3·6	0·6	16·1	1·8	36·2
Specific surface (m^2./g.)	2·3		1·7	
SiO_2 (%)	99·3		99·2	
Ignition loss (%)	0·10		0·57	
Silica solubility (SiO_2 mg./100 ml.)	10·7		9·05	

Anthracite Coal

A low-ash fraction of anthracite coal from the Llandebie Colliery, South Wales, was turbine-ground and a fraction below 10 μ separated by air elutriation with a Roller apparatus. From this material a fraction below 2 μ was prepared by centrifuging in water after adding sufficient detergent ("teepol", Shell) to make the coal wettable. The material below 2 μ equivalent diameter was spun down by prolonged centrifuging, boiled with water to remove as much of the "teepol" as possible, and finally dried. The particle size distribution and the analysis of this dust are given in Table 2.

TABLE 2
ANALYSIS AND PARTICLE SIZE DISTRIBUTION OF ANTHRACITE AND BITUMINOUS COAL

Size in μ	Anthracite Coal		Bituminous Coal	
	Percentage		Percentage	
	By No.	By Mass	By No.	By Mass
0·32–0·45	7·55	0·08	—	—
0·45–0·64	17·30	0·53	4·4	0·37
0·64–0·9	20·71	1·75	18·7	4·26
0·9–1·3	32·89	7·73	49·4	31·2
1·3–1·8	9·26	6·24	23·9	43·4
1·8–2·6	9·26	17·47	3·3	16·76
2·6–3·6	1·46	7·88	0·3	3·99
3·6–5·1	0·78	11·71	—	—
5·1–7·2	0·61	25·92	—	—
7·2–10·2	0·17	20·68	—	—
Specific surface (m^2./g.)	1·9		3·9	
Silica content (%)	1·4		1·05	

Bituminous Coal

A sample of bituminous dust below 2 μ diameter was prepared by applying the same technique to a sample of bituminous coal from the Haig Pit, Cumberland. Its size distribution and analysis are also given in Table 2.

PREPARATION OF DUST SUSPENSIONS

Tridymite

In order to give in 1 ml. of suspension 12·5 mg., 25 mg., and 50 mg. of tridymite, the following amounts were weighed out and shaken with 15 ml. of sterile physiological saline in 25 ml. screw-capped bottles: 0·1875 g., 0·375 g., and 0·75 g. of the tridymite (and 0·375 g. of the electro-dialyzed tridymite to give 25 mg. in 1 ml.). The mixtures were thoroughly shaken for 30 min. in an electric shaking machine, autoclaved for 20 min. at 15 lb. pressure, and then re-shaken until the time of the injection.

Anthracite and Bituminous Coal

Amounts of 0·75 g. anthracite and bituminous coal were weighed and transferred into screw-capped bottles, and 24 ml. sterile physiological saline added to each. The suspensions were thoroughly shaken for half an hour, autoclaved for 20 min. at 15 lb. pressure, and 1 ml. sterile rat serum added to each bottle of suspension to help to disperse and suspend the particles. The final concentration of dust was 30 mg./ml.

Anthracite and Bituminous Coal + Tridymite

Samples each of 0·75 g. of anthracite and of bituminous coal were weighed, and each was mixed intimately with 0·025 g. of tridymite. The anthracite-tridymite and bituminous-tridymite mixtures were made up as above with 24 ml. sterile saline and 1 ml. sterile rat serum. The final concentrations were thus 30 mg. anthracite or bituminous coal and 1 mg. tridymite/1 ml. of suspension.

Tridymite (Control for Coal-tridymite Mixtures)

A suspension of 1 mg. tridymite/ml. of saline was made up by shaking 25 mg. of tridymite with 25 ml. of saline as above.

ANIMALS

The black and white hooded strain of the Medical Research Council was used. They were male rats of average weight 250 g. Ten animals were used in each of the four experiments on tridymite, and 18 were used in each of the five groups with anthracite, bituminous coal, anthracite + tridymite, bituminous + tridymite, and tridymite.

EXPERIMENTAL PROCEDURE

The rats were lightly anaesthetized with ether, the tongue was retracted forward with a small clip and an auroscope fitted with a medium-sized speculum was inserted into the mouth as far as the aurophary nx. The throat was cleaned with a swab and a long, blunt needle (10 cm. × 14 gauge) was passed between the vocal cords into the trachea until it struck the carina, and then it was slightly withdrawn about 2 mm.; 1·1 ml. of dust suspension (about 0·1 ml. usually remains in the long needle and syringe after the injection) was drawn into a 2 ml. syringe from the screw-capped bottles which were kept agitated in a mechanical shaker. The syringe was attached to the long needle and the suspension with 1 ml. of air was quickly and forcibly injected. The needle was withdrawn from the trachea. The animals suffered a short period of apnoea, but they soon regained their normal breathing. There was no regurgitation of

the suspension, and there were no immediate post-operation deaths.

DURATION OF EXPERIMENTS

Tridymite

One rat from the group of animals injected with 12·5 mg., two from the group injected with 25 mg., two from the 25 mg. electro-dialyzed group, and five from that with 50 mg. were lost by death or cannibalism. Because of this larger number of deaths in the last group (50 mg.), it was not possible to obtain equal days of survival in all animals. However, the killing of animals was so adjusted as to enable the lung pathology to be studied till the maximum degree of fibrosis was produced by the different amounts of dust used. With the smallest dose (12·5 mg.) the experiment extended to 300 days, with the 25 mg. doses to 100 days, and with the largest dose (50 mg.) for only 75 days as none of the animals survived beyond that time.

Coal + Tridymite

Of the 18 animals in each of these five groups, several died, and of the rest two from each group were killed at 90, 180, 240, 300, and 330 days. All surviving animals were killed at 350 days.

PATHOLOGICAL TECHNIQUE

Routine post-mortem examinations were carried out on dead and killed animals. Formol-saline (10%) was injected into the lungs before opening the thoracic cavity in animals which were killed, or in those which were found dead the lungs were distended with fixative after their removal from the thoracic cavity. After preliminary fixation blocks were selected through the long axes of both lungs at the level of the hilum, to include the hilar lymph nodes, for histological sections. Blocks were embedded in paraffin and sectioned at 5 μ. Serial sections were stained with haematoxylin and eosin, and by Gordon and Sweets' (1936) silver impregnation method; and micro-incineration was carried out to study the distribution of dust in the lesions. The details of these procedures have been fully recorded in earlier papers in this series. The lymph nodes were dissected and weighed as described by Nagelschmidt, Nelson, King, and Harrison (1954).

COLLAGEN ESTIMATIONS

For the estimations of collagen, the remaining lung tissue from each animal was dried at 105° C., and to it was added the unused tissue from the blocks and trimmings which were dewaxed with xylene. The combined dried tissue of each lung was powdered in an iron mortar, re-dried at 105° C., ground again and carefully mixed.

The collagen estimation depends upon the colorimetric determination of hydroxyproline, the most characteristic amino-acid of collagen and reticulin. The collagen is first transformed into gelatin by autoclaving a sample of the dried tissue. The proteins other than gelatin are precipitated by trichloroacetic acid and filtered off, the gelatin remaining in the filtrate. Hydrolysis of the gelatin in a sample of the filtrate is carried out with hydrochloric acid to liberate the constituent amino-acids, and the hydroxyproline is then determined colorimetrically. The details of this procedure are given by Stacy and King (1954).

RESULTS WITH TRIDYMITE

Gross Appearance of the Lungs

12·5 mg. Tridymite.—Animals in this group showed some scattered subpleural collections of dust in the early stages. These were greyish-white and most of them were seen over the dorsal aspects of the lung. Small discrete areas of fibrosis were seen over both lungs at 90 days, and these appeared larger at 150 days. There was little change from this time until 300 days, when the lung of one animal showed some confluent areas of fibrosis, and the others only patchy fibrosis.

25 mg. Tridymite.—There were subpleural dust collections of well-marked fibrotic areas dorsally at 30 days, and these became confluent by 60 days.

25 mg. Electro-dialyzed Tridymite.—An animal was killed at nine days, and its lungs showed the same subpleural dust collections and a few definite areas of fibrosis. As early as 15 days the fibrotic areas were beginning to become confluent, and by 45 days these confluent areas were larger. A considerable part of the lung tissue was replaced by confluent fibrosis at 75 days, and this was still more marked at 105 days, when all the animals had died or been killed.

50 mg. Tridymite.—Within the first week five animals died. Their lungs were haemorrhagic and parts were consolidated. The animal killed at 15 days had subpleural dust collections dorsally, with discrete areas of fibrosis. The areas of fibrosis were more numerous and more marked at 30 days, and the lungs of the animal dead at 50 days showed confluent fibrosis in both lungs. The last two animals died at 72 days and their lungs showed massive confluent fibrosis with very little lung tissue of normal appearance left.

The tracheo-bronchial lymph nodes in all these groups were enlarged to several times the normal size and were firm, and even hard in the case of animals killed late in the experiments.

Histological Findings

The lesions produced by the various amounts of tridymite were similar and the type of reaction differed only in the degree of severity. The smallest amount (12·5 mg.) produced considerable fibrosis at 30 days, progressing to grade 3 at 60 days, grade 4 at 150 days, and the maximum

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COLLAGEN ESTIMATIONS

For the estimations of collagen, the remaining lung tissue from each animal was dried at 105° C., and to it was added the unused tissue from the blocks and trimmings which were dewaxed with xylene. The combined dried tissue of each lung was powdered in an iron mortar, re-dried at 105° C., ground again and carefully mixed.

The collagen estimation depends upon the colorimetric determination of hydroxyproline, the most characteristic amino-acid of collagen and reticulin. The collagen is first transformed into gelatin by autoclaving a sample of the dried tissue. The proteins other than gelatin are precipitated by trichloroacetic acid and filtered off, the gelatin remaining in the filtrate. Hydrolysis of the gelatin in a sample of the filtrate is carried out with hydrochloric acid to liberate the constituent amino-acids, and the hydroxyproline is then determined colorimetrically. The details of this procedure are given by Stacy and King (1954).

RESULTS WITH TRIDYMITE

Gross Appearance of the Lungs

12·5 mg. Tridymite.—Animals in this group showed some scattered subpleural collections of dust in the early stages. These were greyish-white and most of them were seen over the dorsal aspects of the lung. Small discrete areas of fibrosis were seen over both lungs at 90 days, and these appeared larger at 150 days. There was little change from this time until 300 days, when the lung of one animal showed some confluent areas of fibrosis, and the others only patchy fibrosis.

25 mg. Tridymite.—There were subpleural dust collections of well-marked fibrotic areas dorsally at 30 days, and these became confluent by 60 days.

25 mg. Electro-dialyzed Tridymite.—An animal was killed at nine days, and its lungs showed the same subpleural dust collections and a few definite areas of fibrosis. As early as 15 days the fibrotic areas were beginning to become confluent, and by 45 days these confluent areas were larger. A considerable part of the lung tissue was replaced by confluent fibrosis at 75 days, and this was still more marked at 105 days, when all the animals had died or been killed.

50 mg. Tridymite.—Within the first week five animals died. Their lungs were haemorrhagic and parts were consolidated. The animal killed at 15 days had subpleural dust collections dorsally, with discrete areas of fibrosis. The areas of fibrosis were more numerous and more marked at 30 days, and the lungs of the animal dead at 50 days showed confluent fibrosis in both lungs. The last two animals died at 72 days and their lungs showed massive confluent fibrosis with very little lung tissue of normal appearance left.

The tracheo-bronchial lymph nodes in all these groups were enlarged to several times the normal size and were firm, and even hard in the case of animals killed late in the experiments.

Histological Findings

The lesions produced by the various amounts of tridymite were similar and the type of reaction differed only in the degree of severity. The smallest amount (12·5 mg.) produced considerable fibrosis at 30 days, progressing to grade 3 at 60 days, grade 4 at 150 days, and the maximum

fibrosis (grade 5) at 300 days. The 25 mg. dose produced fibrosis more rapidly, reaching the maximum at 60 days. There was hardly any difference between the reactions produced by the 25 mg. of tridymite, and the same quantity of electro-dialyzed tridymite. The most marked lesions were in the lungs of the animals which received the 50 mg. dose : grade 3 was reached by 30 days and grade 5 fibrosis at 50 days, involving nearly all the lung tissue. In all groups, subpleural drift of dust was seen microscopically, and the pleura overlying the lesions showed thickening. Dust cells were present in some alveoli, in the interstitial tissues and lymphoid follicles. There was marked hyperplasia of the peribronchial lymphoid tissues.

12·5 mg. Tridymite.—At 30 days there was a good dust cell reaction and a marked peribronchial lymphoid reaction. Aggregates of dust cells contained a loose network of fine reticulin fibres (Fig. 1). Only an occasional nodule showed a small amount of collagen. The lesions were nodular and discrete at 60 days and the loose reticulin network was replaced by collagen fibres which were compact (grade 3 fibrosis, Fig. 2). The lesions were larger at 150 days, and almost completely acellular (grade 4 fibrosis). There was little change after this until 300 days, except that some areas of fibrosis became confluent (grade 5).

25 mg. Tridymite.—There was a good dust cell reaction at 30 days, and there were many discrete cellular lesions perivascularly distributed with thick and compact reticulin fibres, and some collagen in many of them (grade 2 fibrosis, Fig. 3). The lesions progressed so rapidly that by 60 days the nodules were mainly acellular, confluent, and collagenous (grade 5 fibrosis, Fig. 4), About half the lung area appeared to be replaced by confluent fibrosis. At 90, 97, and 110 days there was progressively less non-fibrous tissue remaining, and there was some pleural thickening.

25 mg. Electro-dialyzed Tridymite.—The histological picture was very similar to that seen with the 25 mg. of undialyzed tridymite. Reticulin nodules were found as early as nine days (grade 2 fibrosis), and at 15 days nodules were largely acellular and collagenous (grade 3 fibrosis) ; similarly at 30 days. By 45 days the lesions were nearly all confluent, acellular and fully collagenous (grade 5 fibrosis). By the end of the experiment there was very little lung tissue uninvolved by fibrosis, and the pleura was also involved.

50 mg. Tridymite.—During the first week five animals died, and their lungs showed varying degrees of pneumonic consolidation. In the lungs of an animal killed at 15 days there was good dust cell reaction, marked peribronchial lymphoid reaction, and many nodular lesions which were discrete, cellular, and composed of coarse, compact reticulin with a little collagen (grade 2 fibrosis, Fig. 5). At 30 days the nodules were discrete, larger, and somewhat acellular and collagenous (grade 3 fibrosis). At 50 days the right lung of an animal found dead was almost fully composed of large confluent areas of fibrosis which were acellular and fully collagenous (grade 5 fibrosis, Fig. 6). There was more alveolar tissue remaining in the left lung. All remaining animals in this group had died by 72 days, and in all there was almost total replacement and massive confluent fibrosis of the alveolar tissue of both lungs.

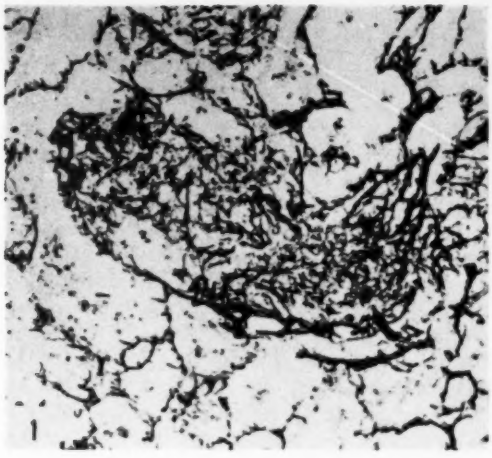
The histological findings in the lungs of the rats receiving the different dosages of tridymite are set out in Table 3 and Fig. 7. The data for 50 mg. of

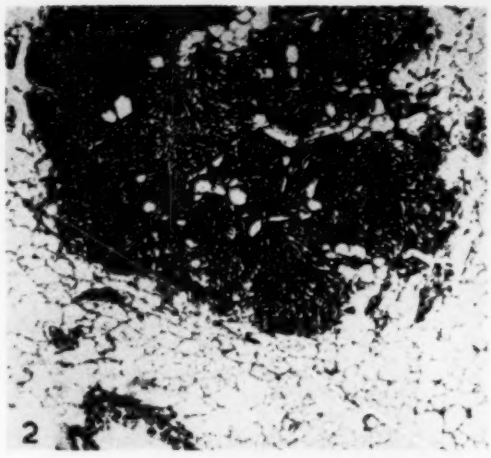
TABLE 3

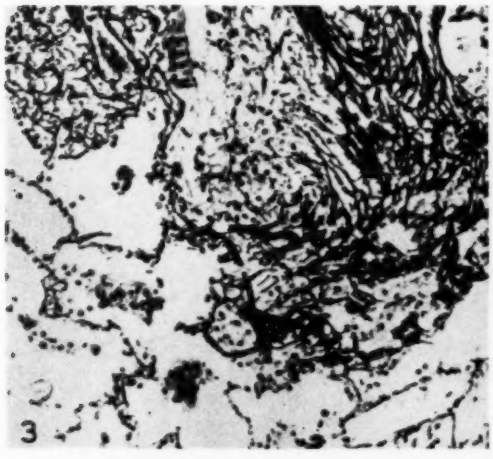
SUMMARY OF PATHOLOGICAL CHANGES IN LUNGS OF RATS PRODUCED BY VARIABLE AMOUNTS OF TRIDYMITE

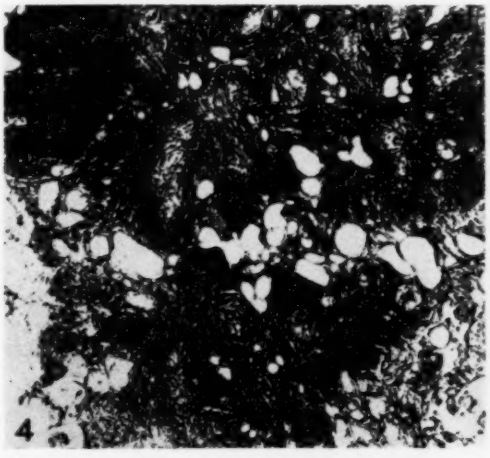
Days of Survival	12·5 mg.		25 mg.		25 mg. (electro-dialyzed)		50 mg.	
	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis
1–9	—	—	Died (1)	—	Killed (1)	2	Died (5)	—
10–15	—	—	Died (1)	—	Killed (1)	2 max.†	Killed (1)	2
16–30	Killed (1)*	1	Killed (1)	2	Killed (1)	3 max.	Killed (1)	3 max.
31–45	—	—	Died (1)	—	Killed (1)	5	—	—
46–60	Killed (1)	3	Killed (1)	5	Killed (1)	5	Died (1)	5
					Eaten (2)			
61–75	—	—	—	—	Killed (1)	5	Died (2)	5
76–90	Killed (1)	3	Killed (1)	5	Killed (1)	5	—	—
91–100	—	—	Died (3)	5	—	—	—	—
101–120	Killed (1)	3	Died (1)	5	Killed (1)	5	—	—
121–180	Killed (2)	4	—	—	—	—	—	—
181–299	Killed (2)	4	—	—	—	—	—	—
	Eaten (1)							
300	Killed (1)	5	—	—	—	—	—	—

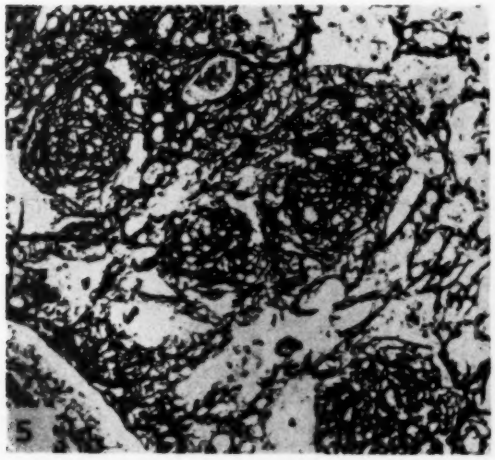
* Numbers in brackets indicate the number of animals killed or found dead during that period. † Max. = Maximal within the grade of fibrosis.











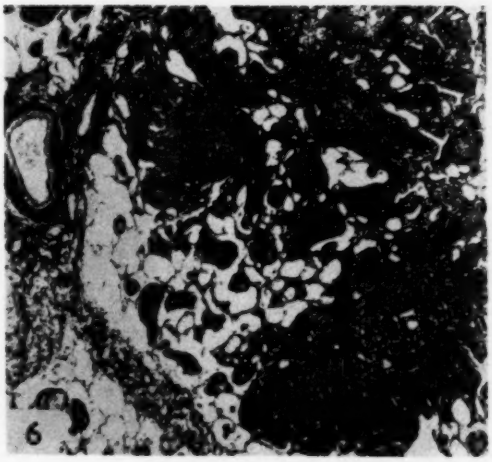


FIG. 1.—Rat lung 30 days after injection of 12·5 mg. tridymite. Nodular lesions showing a loose network of fine reticulin fibres (grade 1 fibrosis). Silver impregnation. ×210.

FIG. 2.—Rat lung 60 days after injection of 12·5 mg. tridymite. Silicotic nodule with thick, compact collagen (grade 3 fibrosis). Silver impregnation. ×65.

FIG. 3.—Rat lung 30 days after injection of 25 mg. tridymite. Nodular lesions with thick and compact reticulin and some collagen (grade 2 fibrosis). Silver impregnation. ×210.

FIG. 4.—Rat lung 60 days after injection of 25 mg. tridymite. Confluent, fully collagenous lesions (grade 5 fibrosis) with a few patent alveoli. Silver impregnation. ×48.

FIG. 5.—Rat lung 15 days after injection of 50 mg. of tridymite. Lesions show thick, compact reticulin and some collagen (grade 2 fibrosis). Silver impregnation. ×210.

FIG. 6.—Rat lung 50 days after injection of 50 mg. tridymite. Confluent lesions with dense collagen (grade 5 fibrosis). A few patent alveoli and blood vessels seen within the lesions. Silver impregnation. ×48.

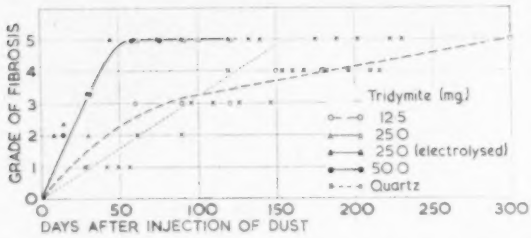
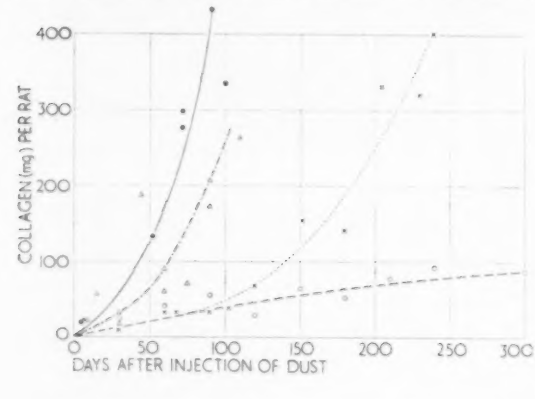


FIG. 7.—Grade of fibrosis of the lungs of rats after injection of different amounts of tridymite and 50 mg. of quartz.

quartz are taken from the controls of the cortisone experiment (Harrison, King, Dale, and Sichel, 1952).

Collagen

Collagen determinations from three of the four groups (omitting the 25 mg. tridymite not electrodialyzed) were made on the lungs of rats killed at monthly intervals. There was an increase with time and with the amount of tridymite administered. These results are set out graphically (Fig. 14). Data from Stacy and King (1954) for 50 mg. of quartz of similar size to the tridymite are added for comparison. As some of the quartz collagen figures were erratic, mean results of 150 and 154 days and of 202 and 210 days were used for plotting. Fig. 14 shows a remarkable increase in the amount of collagen with the largest dose, and a reasonably good relationship between collagen production and tridymite administered up to 70 days when all animals in the 50 mg. tridymite group were dead or killed. The curve for collagen development in the quartz experiment is near that for the 12·5 mg. tridymite group for the first three months, but it rises more steeply later on and reaches after eight months the same high values as 50 mg. tridymite gave after three months.



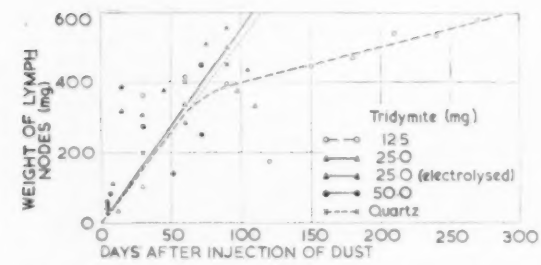


FIG. 14.—Collagen (mg.) in lungs of rats after injection of different amounts of tridymite and 50 mg. of quartz.

FIG. 15.—Weight of lymph nodes of rats after injection of different amounts of tridymite and 50 mg. of quartz.

Development of Lymph Nodes

After injection of quartz or flint, an abnormal swelling of lymph nodes of rats was observed (Nagelschmidt and others, 1954). The fresh weights of the lymph nodes from the four tridymite groups were determined and are shown in comparison with similar results for 50 mg. of quartz in Fig. 15. The quartz data were taken from the controls of the cortisone experiment (Stacy and King, 1954). Although there is a large scatter for individual results, Fig. 15 suggests that for the first 90 days neither amount of tridymite nor type of silica modification has any effect on the results. For longer periods of time the weights of the glands of animals with 12·5 mg. of tridymite were far smaller than those with quartz found by Stacy and King. No animals with higher tridymite doses survived for more than 120 days.

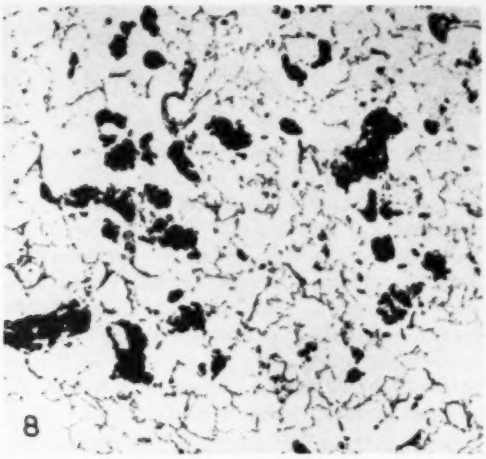
RESULTS WITH COAL + TRIDYMITE

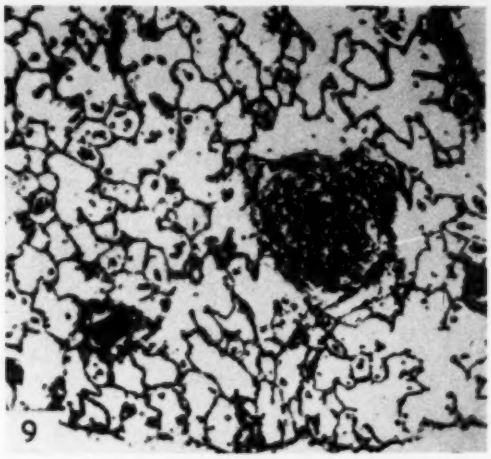
Gross Appearances of the Lungs

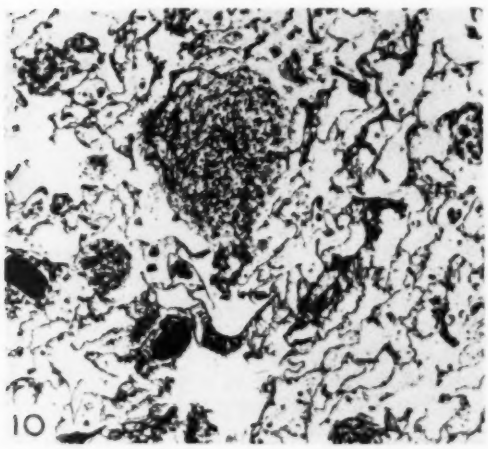
30 mg. Anthracite Coal and 30 mg. Bituminous Coal.—The gross appearances in the two groups were similar. There were collections of dust distributed diffusely over the lung surfaces at 90 days. By 180 days these seemed to be denser ; there were no further changes either at 300 or 350 days. The hilar lymph nodes were enlarged and black.

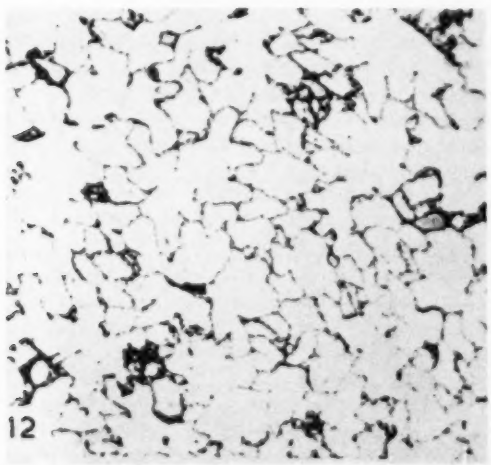
30 mg. Anthracite + 1 mg. Tridymite and 30 mg. Bituminous + 1 mg. Tridymite.—The lungs in these two groups were also very similar. At 90 days there were diffuse deposits of coal dust on the lung surfaces. The collections were larger at 180 days, and small white areas 2 to 3 mm. in diameter were seen associated with them. No other changes, beyond a few pleural adhesions, were visible macroscopically from this time until 350 days. The hilar nodes were somewhat enlarged and black.

1 mg. Tridymite.—In the majority of the lungs there were no visible macroscopic changes at any









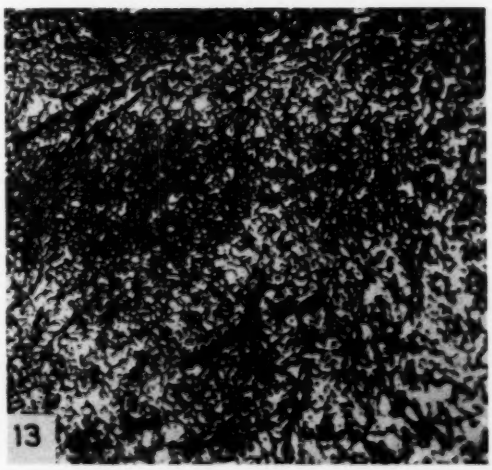


FIG. 8.—Rat lung 90 days after the injection of 30 mg. anthracite coal. Dust gathered into compact nodules without any fibrosis. Silver impregnation. ×120.

FIG. 9.—Rat lung 90 days after the injection of 30 mg. anthracite coal and 1 mg. tridymite. Nodular dust collections with a few fine reticulin fibres arranged loosely (grade 1 fibrosis, min.). Silver impregnation. ×120.

FIG. 10.—Rat lung 90 days after injection of 30 mg. bituminous coal and 1 mg. tridymite. Nodular lesions composed of dust with a loose network of reticulin fibres (grade 1 fibrosis). Silver impregnation. ×120.

FIG. 11.—Rat lung 300 days after injection of 30 mg. bituminous coal and 1 mg. tridymite. Dust lesion adjacent to a blood vessel and containing thick and compact reticulin with some collagen (grade 2 fibrosis). Silver impregnation. ×160.

FIG. 12.—Lung field from same rat as in Fig. 13. 300 days after injection of 1 mg. tridymite. There are no dust collections and there is no fibrosis. Silver impregnation. ×120.

FIG. 13.—Hilar lymph node of same rat as Fig. 12. Loose network of thick reticulin fibres (grade 1 fibrosis max.). Silver impregnation. ×160.

period; but in a few there were small, discrete, white areas scattered over both lung surfaces. The hilar nodes were enlarged and firm.

Histological Findings

30 mg. Anthracite and 30 mg. Bituminous Coal.—The histological appearances of the lungs in these two groups were similar. At 90 days there was some dust lying free in the alveoli; most of it was collected together in the form of irregular foci situated perivascularly. There was no evidence of fibrosis (Fig. 8). A mild degree of emphysema was present around the nodules, and there was hyperplasia of the peribronchial lymphoid tissues with dust in them. There was no change in the histological appearances of the coal dust lesions up to 350 days, except for the dust collections becoming more compact. No fibrosis was seen at any stage, either in the lungs or in the lymph nodes.

30 mg. Anthracite + 1 mg. Tridymite.—At 90 days most of the dust was collected into irregular nodular lesions which were cellular and mainly perivascular. A few were subpleural. A mild degree of focal emphysema was present. The lesions contained loose reticulin fibres (grade 1 fibrosis, Fig. 9). By 180 days the dust lesions were more compact and the reticulin fibres were thicker. There was marked peribronchial fibrosis. There were many irregular, discrete nodules with thick and compact reticulin (grade 2 fibrosis) at 240 days; but there was not much progress beyond this, the lesions remaining within the maximal limits of grade 2 fibrosis at 300 days and at 350 days. The lymph nodes showed only mild reticulinosis.

30 mg. Bituminous + 1 mg. Tridymite.—There was a small amount of dust lying free in the alveoli at 90 days, but the greater part of it was in focal aggregations situated perivascularly. There was a fine network of reticulin fibrils within the lesions (grade 1 fibrosis, Fig. 10). There was marked peribronchial fibrosis and some emphysema. There was no further progress until 240 days, when the nodules appeared to be more compact with thick reticulin fibres (grade 2 fibrosis). There was marked peribronchial fibrosis and some emphysema. By 240 days the nodules appeared to be more compact with thick reticulin fibres (grade 2 fibrosis). The lesions remained within the limits of grade 2 fibrosis at 300 days (Fig. 11), and had not progressed further by 350 days. Mild reticulinosis of the lymph nodes was seen at 300 days and 350 days.

1 mg. Tridymite.—A very few focal aggregations of dust cells were seen in the lungs at 90 days, but there was no fibrosis. By 180 days some peribronchial fibrosis was seen, but there was no fibrosis in the few dust cell collections, although some fibrosis was evident in areas where such lesions were associated with bronchiectatic changes. Apart from this, no fibrosis was found in the lungs at any period up to 350 days.

The hilar lymph nodes contained some dust at 90 days, and by 300 days there were definite lesions which contained a loose network of thick reticulin fibres (grade 1 fibrosis, maximal, Fig. 13), whereas the lungs at this same period revealed no lesions whatever (Fig. 12).

A summary of the pathological changes in the lungs of rats produced by coal and tridymite alone and in combination is given in Table 4.

DISCUSSION

Tridymite

The tridymite used in these experiments produced very rapidly developing fibrosis in the lungs of rats, as compared with other forms of pure silica, which had been used in previous experiments. King and others (1953a) had found that 50 mg. of tridymite produced far more reaction in the lungs

TABLE 4

SUMMARY OF PATHOLOGICAL CHANGES IN THE LUNGS OF RATS PRODUCED BY COAL AND TRIDYMITE ALONE AND IN COMBINATION

Days of Survival	Anthracite 30 mg.		Anthracite 30 mg. + Tridymite 1 mg.		Bituminous 30 mg.		Bituminous 30 mg. + Tridymite 1 mg.		Tridymite 1 mg.	
	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis	Mode of Death	Grade of Fibrosis
1–90	Died (2)*	—	Died (2)	—	—	—	Died (3)	—	Died (1)	—
	Killed (2)	0	Killed (2)	1 min.†	Killed (2)	0	Killed (2)	1	Killed (2)	0
91–180	—	—	Died (4)	—	Died (1)	—	Died (1)	—	—	—
	Killed (2)	0	Killed (2)	1	Killed (3)	0	Killed (2)	1	Killed (2)	0
181–240	Died (1)	—	Died (1)	—	—	—	—	—	—	—
	Killed (3)	0	Killed (2)	2	Killed (2)	0	Killed (2)	2 min.†	Killed (2)	0
241–300	—	—	Died (1)	—	—	—	—	—	—	—
	Killed (2)	0	Killed (2)	2	Killed (2)	0	Killed (2)	2	Killed (2)	0
301–350	Killed (8)	0	Died (1)	—	—	—	Died (1)	—	—	—
	—	—	Killed (1)	2	Killed (8)	0	Killed (5)	2	Killed (9)	0

* Numbers in brackets indicate the number of animals killed or found dead during that period. † Min. = Minimal within the stated grade.

of rats than the same amount of the other forms of pure silica—amorphous vitreous silica, quartz, and cristobalite. All these silica modifications were of the same high purity, *i.e.*, about 99% SiO_2, the same particle size, and silica solubility. In that experiment tridymite produced fully collagenous confluent fibrosis in 60 days as compared with 270 days for quartz. In the present experiments the same dose of tridymite, 50 mg., acted similarly and produced the same degree and amount of fibrosis in about the same time, *i.e.*, 50 days. A quarter of the amount (12·5 mg.) produced similar lesions in 300 days, which is a little longer than the time taken by 50 mg. of quartz.

We had thought that the results of the previous animal tests with tridymite might partly be due to the presence of traces of hydrofluoric acid, although we could not demonstrate this chemically. The dust used here had never been in contact with hydrofluoric acid. Nevertheless, it seemed advisable to subject a sample of it to electro-dialysis to make certain of the elimination of any possible adsorbed ions. The severity of the fibrosis was not lessened by this treatment, and there seems no doubt that the extremely rapid and severe fibrosis is due to tridymite itself.

The results of the collagen estimations confirm the conclusions of the histological examinations, and give the possibility of an added, quantitative assessment of fibrosis, which it has not been possible satisfactorily to achieve by histological means. The histology of silicosis is essentially a qualitative matter. It reveals the nature of the lesions, the distribution and progress of the fibrosis and its severity, but it gives only a rough idea of the total amount of fibrosis present in a tissue. The collagen estimations, in conjunction with the histological examinations, have the advantage of enabling us to say not only what sort of fibrosis is present, but how much of it there is. The curves in Fig. 14 show that collagen production takes place at a steady rate with the 12·5 mg. dose of tridymite. Approximately 0·8 to 1·2 mg. of collagen is produced per month per mg. of tridymite, which has 23 cm^2. of surface.

The curves for quartz and for the higher tridymite levels do not appear to be straight lines. The data are not sufficient in number and they are too erratic to warrant a detailed discussion, but they suggest that the rate of collagen production for both higher tridymite levels is the same and is about twice as high as that for the lowest level.

Fifty milligrams of quartz appear to produce only 0·34 mg. of collagen per mg. of quartz per month during the first four months, but the rate is nearly five times as high, namely 1·6 mg. of collagen, for the next four months. Further work is required to confirm these early impressions.

The reason for the higher rate of collagen production by tridymite as compared with quartz is still entirely unknown. The difference does not appear to be related to silica solubility and this leaves surface characteristics or crystal structure as possible alternatives.

Coal + Tridymite

The results with anthracite and bituminous coal dusts confirm those of previous experiments that no fibrosis was produced by coal dust in the lungs of animals. The only histological change observed during the course of the experiment was a gradual sweeping of the injected coal particles into focal accumulations of dust deposits. The 1 mg. of tridymite used alone likewise caused no fibrosis in the lungs. Pulmonary infection was found in the lungs of one animal which died, and there was fibrosis associated with the infective lesions. But the results in the remaining animals were uncomplicated by infection, and no lesions were demonstrated in the lungs of any of the animals, although some fibrosis was demonstrated in the hilar lymph nodes at 300 days and 350 days. This is probably due to the small amount of tridymite dust being removed from the lungs to the lymph nodes before any pulmonary fibrosis occurs. Ray and others (1951) obtained similar results with 2 mg. of quartz.

When the same quantity of tridymite (1 mg.) was injected together with anthracite or bituminous coal in 30 mg. doses, definite pulmonary lesions were produced. Reticulin fibrosis of grade 1 was observed at 90 days, and of grade 2 by 240 days in both groups. The lesions advanced only slightly beyond this stage, remaining within the maximal limits of grade 2 at 300 and 350 days. It is likely that the inert coal retards the movement of tridymite in the lymphatics causing it to be retained in the lungs where it exerts a fibrogenic effect. With a much higher percentage (20%) of quartz used with coal-mine dust Belt and King (1945) observed grade 4 fibrosis at 365 days, and Ray and others (1951) grade 3 fibrosis when 2 mg. of quartz was used with 98 mg. of coal. The smaller amounts of tridymite (1 mg.) and coal (30 mg.) used in these experiments produced grade 2 fibrosis when injected in combination.

No differences were observed between anthracite and bituminous coal when used with tridymite or alone. Neither coal appeared to act as an antidotal dust when used with free silica in animals. On the other hand they appeared to enhance the effect of small amounts of silica by keeping it in the lungs.

SUMMARY

Different amounts of pure tridymite dust of equal particle size, injected into the lungs of rats, produced the maximum degree of fibrosis in 300 days with 12·5 mg. of tridymite, in 60 to 70 days with 25 mg., and in 50 days with 50 mg. With the 50 mg. dose most of the lung tissue was replaced by fibrous areas. Chemical determinations of collagen demonstrated both a more rapid and a larger total production of fibrous tissue with the larger doses of tridymite. Tridymite, 12·5 mg., caused, during the first three months, the production of as much collagen as 50 mg. of quartz, but produced after eight months only one quarter of the amount which 50 mg. of quartz gave. The quartz had then produced as much collagen as 50 mg. of tridymite in three months.

Mixtures of anthracite and bituminous coal dust with a small amount of tridymite (1 mg.) produced lesions of grade 2 fibrosis in the lungs. Anthracite and bituminous coal by themselves did not produce any fibrosis in lungs or hilar lymph nodes in 350 days. Tridymite, 1 mg., appeared to be completely eliminated from the lungs into the hilar lymph nodes: fibrosis was observed in the lymph nodes, but none was found in the lungs.

The authors are indebted to the Medical Research Council for a grant towards the expenses of the investigation, to Mr. E. H. M. Badger, of the North Thames Gas Board laboratory, for supplying the tridymite, to Mr. B. D. Stacy for the collagen determinations, to Mr. J. Cartwright for the size distributions, to Mr. B. C. S. Hollands for technical help with the animals and in the cutting and staining of sections, and to the Ministry of Fuel and Power for permission to publish.

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TOXICOLOGICAL INVESTIGATIONS ON DIMETHYLFORMAMIDE

BY

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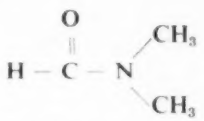
From the Akademie für Sozialhygiene, Arbeitshygiene und ärztliche Fortbildung, Berlin-Lichtenberg

(RECEIVED FOR PUBLICATION JUNE 24, 1955)

Dimethylformamide is the only solvent from which it is possible to spin the polymer polyacrylonitrile for the production of "orlon" fibre. Because nothing was known of its biological properties and workers making polyacrylonitrile fibres come into contact with it, an examination of its toxicity was undertaken.

Chemical Properties

Dimethylformamide is a colourless liquid with the formula :—



The boiling point is 153° C., and it is miscible with water and some organic solvents. On boiling with water or alkali it hydrolyzes to formic acid and dimethylamine. It is more stable than formamide and can be distilled. For the present work freshly distilled dimethylformamide was used and all aqueous solutions were freshly prepared.

Dimethylformamide in the atmosphere may be trapped by passing the air through two bubblers containing water and estimating it by the method of Bergmann (1952). Of a dimethylformamide solution, 10 ml. was mixed with 1 ml. 3·5 N-NaOH and 1 ml. 2N-hydroxylamine sulphate. After shaking, it was left at 15° C. for four hours and 1 ml. of 3·5 N-HCl and 0·74 M-$FeCl_3$ added and the colour estimated. It may be estimated directly in blood after precipitating the proteins or in filtrates of urine or stomach contents.

Toxicology

Acute Toxicity.—The L.D.50 was determined by the method of Kärber (1931) using seven animals per dose. Albino mice (18 to 23 g.), albino rats (180 to 220 g.), rabbits, and cats were used after 12 hours' starvation. They were observed for 21 days. Using 49 mice given intraperitoneal injections, the L.D.50 was found to be 0·3 ml. dimethylformamide/100 g. The greatest tolerated dose was 0·05 ml. and the certain lethal dose (L.D.100) 0·6 ml.

The results on rats are presented in Table 1. Over a 24-hour observation period the L.D.50 for rats by subcutaneous injection was 0·78 ml./100 g.

TABLE 1

THE ACUTE TOXICITY OF DIMETHYLFORMAMIDE TO RATS

Route	No. of Animals	Maximum Tolerated	L.D.50	L.D.100	Survival Time
Intraperitoneal	42	0·01	0·14	0·40	6 hr.–10 days
Subcutaneous	56	0·10	0·38	0·80	6 hr.–6 days
Oral	56	0·10	0·37	0·80	12 hr.–12 days

Dose ml./100 g. body weight.

After the injection some animals appeared to be anaesthetized and then died rapidly or recovered in less than two hours to die later. Apart from some general apathy the animals showed little else except a loss of appetite which was sometimes complete and an accompanying loss of weight. Tremors, convulsions, and paralyses were never seen.

The results on cats and rabbits injected intraperitoneally are presented in Table 2.

TABLE 2

THE ACUTE TOXICITY OF DIMETHYLFORMAMIDE GIVEN INTRAPERITONEALLY TO CATS AND RABBITS

Species	Dose (ml. kg.)	No.	Deaths	Survival Time (Days)
Cat	0·25	1	0	—
	0·3	1	0	—
	0·5	1	1	15
	1·0	2	2	5–16
	2·0	1	1	9
Rabbit ..	1·0	4	2	4
	2·0	4	4	3–12

Groups of seven fish (*Rhodeus amarus*) were placed in solutions of dimethylformamide for 24 hours and then put back into freshly aerated water and observed for 21 days. A 0·1% solution of dimethylformamide had no effect; 0·5% dimethylformamide had no immediate effect but two fish died during the next 15 days. In 1% dimethylformamide the fish were slightly uncoordinated after 30 minutes. After two hours two fish lay on their sides and one died after four hours. After eight days five of the six survivors had died.

Skin Absorption.—Rats' tails were dipped in concentrated dimethylformamide solutions at 40° C. for eight hours. Considering the tails as cylinders the area exposed was calculated to be 13·9 to 14·2 cm². of skin for the rats of 160 to 220 g. The tails were rinsed after exposure but became tanned and mummified in a few days. Groups of seven rats were exposed to 25, 50, 75, and 100% concentrations. All those exposed to 100% dimethylformamide died in one to four days after losing 10 to 30 g. body weight. Two of the seven rats exposed to 75% died. The others lost weight during the first few days and then regained it. None of the other rats died. It was calculated that at least 0·8 ml. of 100% dimethylformamide had been absorbed through 14 cm². of skin.

Pathological Findings.—No changes in the red or white blood cells of cats and rabbits were observed and the sternal marrow was normal. In one rabbit and one cat which died the haemoglobin fell.

The blood sugar level in cats and rabbits rose after the intraperitoneal injection of dimethylformamide and hyperglycaemia might be maintained for up to 24 hours (Fig. 1). Glucose tolerance tests on rabbits were normal.

The urines of both cats and rabbits were acid, and during the hyperglycaemic phase sugar appeared in the urine. The urine deposit was normal and albuminuria was not observed. The E.C.G. of poisoned cats was abnormal for several days or until death.

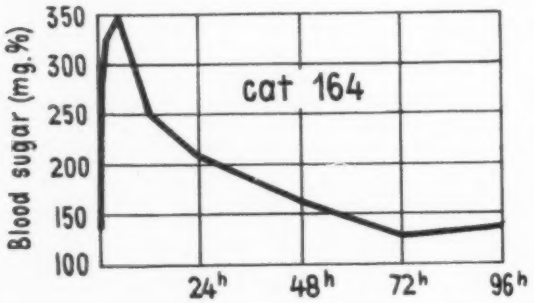


FIG. 1.—The blood sugar curve of a cat injected with 2 ml. dimethylformamide/kg.

Necropsies were performed on all animals that died and of every group the organs of three were examined histologically. The most striking and consistent lesions were in the liver which in rabbits and cats was reduced in size and clay coloured. In six of eight animals examined there was fatty degeneration of the liver cells with widespread necroses (Figs. 2 and 3). The stomach, lungs, heart, spleen, kidney, suprarenals, pancreas, and brain showed no consistent changes.

Chronic Toxicity.—Groups of two cats and 16 rats were put in a gas-tight chamber (0·6 m³.) for eight hours daily except Sundays for up to 120 days. Ventilation was at the rate of 180 to 200 l. /hr. and in three experiments the air contained 100, 230, and 450 p.p.m. dimethylformamide. Two analyses were carried out each day. Each week the animals were weighed and every two weeks the blood was examined, an E.C.G. taken, and liver function tests carried out on the cats.

At the highest concentrations of dimethylformamide the cats ate badly and lost weight but no other signs were observed. All the rats grew normally and appeared unaffected.

Some rats were killed during the experiment and at the end all the cats and three more rats were examined. An irregular incidence of liver necrosis similar to that seen in the acute experiments was observed in the rats despite their normal outward appearance. In the cats fatty degeneration but no necrosis was seen in the livers.

Bronchopneumonic changes were observed in some animals. Hyperaemia of the brain, cloudy swelling of the uriniferous tubules of the kidney, and iron deposits in the spleen were also recorded. During life the blood picture, urine, and E.C.G. remained normal.

Metabolism of Dimethylformamide.—Repeated small doses of dimethylformamide were injected every three minutes intravenously into fasting anaesthetized cats until a total of 6 ml. had been given. Previously the trachea had been cannulated and the pylorus of the stomach tied. All expired air was passed through a bubbler to trap the dimethylformamide. Two hours after the last injection the cats were killed. The stomach contents, expired air, and urine formed were analysed for dimethylformamide and also for formic acid and dimethylamine. Neither of the last two was detected. The total quantities of dimethylformamide recovered are given in Table 3.

FIG. 2.—The liver of a rat two days after 0·15 ml.dimethylformamide/100 g. intraperitoneally. Extensive haemorrhages with necroses of the remaining liver cells. × 19.

FIG. 3.—The same liver starved for fat showing the sharp line of demarcation between surviving and necrotic liver with the cells containing fat. × 125.

TABLE 3

DIMETHYLFORMAMIDE RECOVERED FROM CATS AFTER REPEATED INTRAVENOUS INJECTION OF SMALL DOSES TO A TOTAL OF 6 ML.

Cat Weight (kg.)	Dimethylformamide (mg.) Recovered			
	Urine	100 ml. Blood	Stomach	Expired Air
2·9	16·5	3·7	6·0	—
3·8	12·8	3·1	7·8	0·1
4·7	8·9	5·0	9·2	0·04

Within 24 hours of a dose of 1 to 2 ml. dimethylformamide/kg. body weight rabbits excreted 10 to 15% in the urine.

Action on Skin and Mucous Membranes.—Applied for 24 hours on a soaked rag or painted daily for 28 days, dimethylformamide had no more than a transient slightly irritant effect on human skin. Instilled into the conjunctival sac of the rabbit a 25% solution had no effect; 50% was slightly irritant and 75 to 100% produced a more severe inflammation.

Discussion

These experiments show dimethylformamide to be of intermediate toxicity by comparison with other solvents in common use. The toxic effects are similar in all the species tested though fish seem surprisingly resistant. The reactions of repeated exposure by inhalation are essentially the same as those seen in acute poisoning.

The most striking lesion is the damage to the liver with an associated loss of weight and anorexia. The rise in blood sugar seen in the early stages of poisoning cannot readily be explained. There is probably no primary disturbance of the carbohydrate metabolism and the pancreas shows no histological evidence of damage. Similar changes in blood sugar may be seen in CO poisoning (Moeschlin, 1939) and toluene poisoning (Dimitrenko, 1954).

The changes in the E.C.G. and the deposition of iron in the spleen are probably non-specific and may be observed in animals poisoned by a number of different substances.

The damage to the kidney seen in chronically poisoned animals might be primary or a secondary sign of general poisoning. There is a similarity between the chemical and toxicological properties of dimethylformamide and dimethylnitrosamine. The latter substance also causes widespread liver

necrosis in laboratory animals (Barnes and Magee, 1954). But haemorrhages in the gut and peritoneum were observed only in animals poisoned with dimethylnitrosamine. Carbon tetrachloride also damages both the liver and kidney. It is impossible to say whether the similarity between the action of carbon tetrachloride and dimethylformamide is purely one of chance or that it can be explained by a similarity in their metabolism and mode of action in the body.

Summary

Dimethylformamide is used as a solvent for polyacrylonitrile. It is moderately toxic to rats, mice, rabbits, and cats with an L.D.50 of 0·14 to 0·37 ml./100 g. body weight for rats, depending on the route of application. Death took place up to 12 days after a single dose and liver damage is the most prominent feature with some accompanying kidney damage. Dimethylformamide does not irritate the skin or mucous membranes but can be absorbed through the intact skin of the rat. Some is excreted unchanged in the urine.

Repeated doses by inhalation showed rats to be unaffected by concentrations up to 420 p.p.m., while cats were upset by concentrations of 100 p.p.m.

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Brit. J. industr. Med., 1956, **13**, 55.

DERMATITIS FROM MANSONIA WOOD

BY

L. B. BOURNE

From Cossor House, London

(RECEIVED FOR PUBLICATION JUNE 16, 1955)

Horner and Wigley (1936) reported a case of dermatitis due to Mansonia wood (*Sterculiacea altissima*) and in his annual report the Chief Inspector of Factories (1936) also reported that the dust of this wood could give rise to serious symptoms and in this respect compared it with African box wood.

There are apparently no other reports of the possible dangers relating to the use of this wood, and, as it has been imported in recent years in much larger quantities than hitherto, an account of an outbreak of dermatitis is likely to be of interest as other outbreaks are known to have occurred elsewhere in this country. The wood itself is exported from the Gold Coast and Nigeria and is used locally for general carpentry and in railway coaches. As it resembles black walnut it has been used for piano cases, furniture, shop fittings, and camera boxes. It has been found very useful as a substitute for true walnut with which it should not be confused. In the fresh state the wood is dark brown but on exposure changes to light brown. It air-seasons readily and has considerable natural durability. It was recently found to be ideal for use in the manufacture of radio and television cabinets and the outbreak reported here took place in a factory which makes these products.

All the men affected worked in one small department although the wood in its various stages through the factory was handled by many other workers. On investigation the men in the department concerned were those engaged in fine sanding, whereas in the rest of the factory, although the same wood was handled by many other workers, no men appeared to be involved.

The wood enters the factory in planks which may vary in colour from light brown to a darkish grey brown, the bark remaining much paler. It is then rough sawn and planed in the mill and the particles of reasonably large size are thrown off and extracted by an efficient exhaust system. The work of assembly into cabinets also appears to carry no risk as at no time have there been complaints. It is only when the cabinets are taken to the fine sanding department that difficulties arise. Here the wood is treated by hand sanding with fine carborundum type papers to smooth the surface for further treatment with stains and lacquers. This department has an efficient ventilation system proved to be satisfactory by years of experience with wood dusts of all types. The fine powder given off during the sand papering operation has been shown to be the direct cause of the symptoms.

Twelve men worked in the department, many of whom had been there for some years using many types of wood, but this was the first occasion on which any of them had complained. Six of the men were markedly affected and one only slightly.

The first symptoms were those of nasal irritation and sneezing followed by itching which increased during the working week but diminished considerably during the week-end. If prolonged contact with the dust was maintained there followed increased irritation which spread from the exposed areas of the skin. This was followed by sores in the nostrils, discharge from the nose and epistaxis, and in some cases discharge from the external auditory meatus. Nausea was not marked and there were no complaints of weakness of the arms or legs as previously described by Horner and Wigley (1936).

The dermatitis is characterized by an erythema which is followed by a folliculitis with exudation and some exfoliation. Occasionally there is considerable oedema of the face particularly in the orbital regions (Fig. 1). The fine powder settles on the head and, when the hair is combed, gives rise to an irritation of the scalp, the neck, and between the shoulder blades. It also may settle inside the clothing anywhere but does so particularly in the socks (Fig. 2). Sore throats are common.

It was interesting to note that during the annual holiday period all those involved improved very much and when they returned to work after two weeks' rest they were practically symptom free. There was an immediate recurrence once the work

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FIG. 1.—*Case F. H.* Erythema of face and forehead. Periorbital oedema and ulceration of left eyelid.

FIG. 2.—*Same case.* Typical dermatitis from dust inside sock.

was restarted. Protective masks (Martindale type) and barrier creams were supplied but although these did reduce the nasal symptoms and protect the hands, the rest of the exposed parts of the face, ears, and scalp continued to be affected. Patch tests of six workers gave a marked result in eight to 24 hours with an erythematous itching area which persisted for as long as four days, becoming indurated at the end of this period and gradually fading after some weeks.

The method of testing was as follows :—

An area about 2 in. square of both forearms on the flexor surface was cleaned with ether and allowed to dry. Some Mansonia dust was put on sterile gauze and this was applied to one arm and covered with sticking plaster. This procedure was repeated on the other arm without any dust. A further control was carried out on the author using a similar procedure. Local irritation was felt in about 12 hours and a marked erythema in 24 hours ; the offending powder was then removed. The powder appeared to be unchanged but the skin affected was erythematous, slightly raised, and had to be treated with antipruritics for about 10 days.

It was decided to stop using the wood and all those workers who have remained in the employ of the factory have recovered, are symptom free and are not affected by the other woods in general use, the majority being imported, some of tropical origin.

Two workers left the employ of the company and no further records are available.

Case Histories

Case 1.—F. H., aged 31, a woodworker, began work at the factory on January 2, 1950. On March 5, 1953, he complained of itching fingers after using "special" wood for some days but had no obvious signs. The itching subsided at the week-end but began again within a few hours of using the wood. He did not again use the wood until July 20 and on July 23 had itching fingers with erythema of the backs of the hands and forearms. On August 10, 1953, he returned to work after a holiday with a clear skin. On August 12 he complained of irritation of the nose and itching hands. By the next day the irritation of the hands and forearms had increased, he had a discharge from the ears, superficial ulceration of the nostrils, and marked erythema of the face and scalp. He was taken off Mansonia wood.

On August 21 a patch test of the arms with Mansonia wood powder gave a marked reaction in 12 hours. By August 25 the patch area—indurated, erythematous, tingling—was still marked but the involved areas of the face, arms, and nose were clearing. By December 15 the skin was clear and the other symptoms had subsided.

On May 3, 1954, he used Mansonia wood for a small job and within a few minutes tingling began.

Case 2.—H. P., aged 47, a woodworker, began work on September 27, 1948. From March 9 to 16, 1953, he was absent with a certificate of "acute coryza" after using the wood for some days. On April 20 he had an itching erythema on both arms and face after a few days' exposure. On April 22 a patch test gave a marked positive reaction in 24 hours, and the patient was taken off the work. He has had no trouble since.

Case 3.—J. W., aged 34, a woodworker, began work on March 18, 1953. On May 27 he had an irritation on the left shin, which he stated had been there for some time, clearing up and recurring. It cleared up with lin. cal.

On August 18 the leg was clear but the patient now complained of irritation of the face and between the fingers. He also had oedema and erythema of the upper lip and cheeks, the periocular areas, and the brow. He was taken off the work and the skin cleared. On April 22 a patch test had given a marked positive reaction in 24 hours.

Case 4.—D. A., aged 33, a woodworker, worked at this factory from September 29, 1948, to January 3, 1952, and then again from January 9, 1953. On October 5 he had a cold with rhinitis which persisted and erythema of the hands and face which was not reported. He was off work. On October 27 he reported with dermatitis of the ears, neck, ankles, and axillae. On October 26 a patch test had given a marked positive reaction in eight hours. By the morning of December 15 the skin was clear. In the afternoon of that day he was further exposed to dust for half an hour, and the erythema and pruritus recurred. On March 24 he was exposed to Mansonia wood in error and symptoms returned.

Case 5.—H. Y., aged 47, a woodworker, began work in February, 1953. On October 7 he started to use Mansonia wood and in two days had an irritation of the face, arms, and hands. This was relieved overnight but began again next morning at work. This sequence continued for two weeks. On October 23 he reported to the surgery with follicular erythema of both arms and exudative dermatitis of the cubital fossae, erythema of the face, and follicular dermatitis on the centre of the back between the scapulae. A patch test on October 26 gave a marked positive reaction in 12 hours. By December 15 the skin was clear.

No worker gave a history of previous skin involvement and all had been examined for skin disease before starting work. All the workers had previous experience with many woods for periods varying from 14 to 33 years. The dates of the first exposure to the wood vary because after the first outbreak in March, 1953, the work was re-routed through the factory at different times in order to check the effect of the wood on different operatives. The department in which fine sanding was carried out was separate and did not form part of the rest of the factory, and the incidence of colds and upper respiratory tract affections was no higher there than in any other part of the factory in previous years.

Discussion

The Chief Inspector's report quoted states that "an analysis on Mansonia wood is being done and it is possible that an alkaloid similar to that present in African box wood will be found".

The Officer in Charge, Chemistry Section of The Forest Products Research Laboratory, states:

> "We have had other reports that the fine dust produced in working this timber is liable to cause irritation to the eyes, nose, and throat and there have been some cases of dermatitis attributed to this cause. . . . Information on the chemical components of woods responsible for their toxic or irritant effects is very scanty. In the case of Mansonia nothing appears to be known about the chemical nature of the minor components of the wood, and I do not think it can be assumed that the offending substance is an alkaloid unless direct evidence is available on this point. We have investigated one or two timbers here with the object of obtaining information on the types of component which produce these toxic effects, but with rather indefinite results. Thus the Trinidad timber toporite (*Hernandia sonora*), which causes skin irritation, contains the acetate of podophyllotoxin, which might be expected to be the responsible component, but the medical tests we had carried out were inconclusive.
>
> "In a recent paper Sandermann and Barghoorn report that lapachonone isolated from the South American timber peroba de campos (*Paratecoma peroba*) has irritant properties, although it had been stated earlier that the toxicity of this wood was due to resin acids in it. I mention these examples merely to emphasize the discrepancies and the lack of well-established data which prevail in this field. You will notice that neither podophyllotoxin nor lapachonone is an alkaloid.
>
> "The chemical examination of wood components is rather a lengthy process. It is necessary to extract the wood with one or more solvents, *e.g.*, water, alcohol, ether, fractionate the extracts further by some suitable method, and examine the various fractions for their physiological effects. If they produce positive effects they must be further purified, for they are generally complex mixtures, and characterized chemically. I do not think there is any effective short cut to this kind of investigation."

Schwartz, Tulipan, and Peck (1947) state:

> "Wood from foreign localities or woods not handled before are the ones that usually cause dermatitis".

The workers become affected several days after beginning to work with the new wood. It is usually a sensitization dermatitis, only a few of the workers being affected. The majority of those affected become "hardened" and develop a hyposensitization after they recover. A small percentage of those affected remain sensitized.

FIG. 1.—*Case F. H.* Erythema of face and forehead. Periorbital oedema and ulceration of left eyelid.

FIG. 2.—*Same case.* Typical dermatitis from dust inside sock.

was restarted. Protective masks (Martindale type) and barrier creams were supplied but although these did reduce the nasal symptoms and protect the hands, the rest of the exposed parts of the face, ears, and scalp continued to be affected. Patch tests of six workers gave a marked result in eight to 24 hours with an erythematous itching area which persisted for as long as four days, becoming indurated at the end of this period and gradually fading after some weeks.

The method of testing was as follows :—

An area about 2 in. square of both forearms on the flexor surface was cleaned with ether and allowed to dry. Some Mansonia dust was put on sterile gauze and this was applied to one arm and covered with sticking plaster. This procedure was repeated on the other arm without any dust. A further control was carried out on the author using a similar procedure. Local irritation was felt in about 12 hours and a marked erythema in 24 hours ; the offending powder was then removed. The powder appeared to be unchanged but the skin affected was erythematous, slightly raised, and had to be treated with antipruritics for about 10 days.

It was decided to stop using the wood and all those workers who have remained in the employ of the factory have recovered, are symptom free and are not affected by the other woods in general use, the majority being imported, some of tropical origin.

Two workers left the employ of the company and no further records are available.

Case Histories

Case 1.—F. H., aged 31, a woodworker, began work at the factory on January 2, 1950. On March 5, 1953, he complained of itching fingers after using " special " wood for some days but had no obvious signs. The itching subsided at the week-end but began again within a few hours of using the wood. He did not again use the wood until July 20 and on July 23 had itching fingers with erythema of the backs of the hands and forearms. On August 10, 1953, he returned to work after a holiday with a clear skin. On August 12 he complained of irritation of the nose and itching hands. By the next day the irritation of the hands and forearms had increased, he had a discharge from the ears, superficial ulceration of the nostrils, and marked erythema of the face and scalp. He was taken off Mansonia wood.

On August 21 a patch test of the arms with Mansonia wood powder gave a marked reaction in 12 hours. By August 25 the patch area—indurated, erythematous, tingling—was still marked but the involved areas of the face, arms, and nose were clearing. By December 15 the skin was clear and the other symptoms had subsided.

On May 3, 1954, he used Mansonia wood for a small job and within a few minutes tingling began.

Case 2.—H. P., aged 47, a woodworker, began work on September 27, 1948. From March 9 to 16, 1953, he was absent with a certificate of "acute coryza" after using the wood for some days. On April 20 he had an itching erythema on both arms and face after a few days' exposure. On April 22 a patch test gave a marked positive reaction in 24 hours, and the patient was taken off the work. He has had no trouble since.

Case 3.—J. W., aged 34, a woodworker, began work on March 18, 1953. On May 27 he had an irritation on the left shin, which he stated had been there for some time, clearing up and recurring. It cleared up with lin. cal.

On August 18 the leg was clear but the patient now complained of irritation of the face and between the fingers. He also had oedema and erythema of the upper lip and cheeks, the periocular areas, and the brow. He was taken off the work and the skin cleared. On April 22 a patch test had given a marked positive reaction in 24 hours.

Case 4.—D. A., aged 33, a woodworker, worked at this factory from September 29, 1948, to January 3, 1952, and then again from January 9, 1953. On October 5 he had a cold with rhinitis which persisted and erythema of the hands and face which was not reported. He was off work. On October 27 he reported with dermatitis of the ears, neck, ankles, and axillae. On October 26 a patch test had given a marked positive reaction in eight hours. By the morning of December 15 the skin was clear. In the afternoon of that day he was further exposed to dust for half an hour, and the erythema and pruritus recurred. On March 24 he was exposed to Mansonia wood in error and symptoms returned.

Case 5.—H. Y., aged 47, a woodworker, began work in February, 1953. On October 7 he started to use Mansonia wood and in two days had an irritation of the face, arms, and hands. This was relieved overnight but began again next morning at work. This sequence continued for two weeks. On October 23 he reported to the surgery with follicular erythema of both arms and exudative dermatitis of the cubital fossae, erythema of the face, and follicular dermatitis on the centre of the back between the scapulae. A patch test on October 26 gave a marked positive reaction in 12 hours. By December 15 the skin was clear.

No worker gave a history of previous skin involvement and all had been examined for skin disease before starting work. All the workers had previous experience with many woods for periods varying from 14 to 33 years. The dates of the first exposure to the wood vary because after the first outbreak in March, 1953, the work was re-routed through the factory at different times in order to check the effect of the wood on different operatives. The department in which fine sanding was carried out was separate and did not form part of the rest of the factory, and the incidence of colds and upper respiratory tract affections was no higher there than in any other part of the factory in previous years.

Discussion

The Chief Inspector's report quoted states that "an analysis on Mansonia wood is being done and it is possible that an alkaloid similar to that present in African box wood will be found".

The Officer in Charge, Chemistry Section of The Forest Products Research Laboratory, states:

> "We have had other reports that the fine dust produced in working this timber is liable to cause irritation to the eyes, nose, and throat and there have been some cases of dermatitis attributed to this cause. . . . Information on the chemical components of woods responsible for their toxic or irritant effects is very scanty. In the case of Mansonia nothing appears to be known about the chemical nature of the minor components of the wood, and I do not think it can be assumed that the offending substance is an alkaloid unless direct evidence is available on this point. We have investigated one or two timbers here with the object of obtaining information on the types of component which produce these toxic effects, but with rather indefinite results. Thus the Trinidad timber toporite (*Hernandia sonora*), which causes skin irritation, contains the acetate of podophyllotoxin, which might be expected to be the responsible component, but the medical tests we had carried out were inconclusive.
>
> "In a recent paper Sandermann and Barghoorn report that lapachonone isolated from the South American timber peroba de campos (*Paratecoma peroba*) has irritant properties, although it had been stated earlier that the toxicity of this wood was due to resin acids in it. I mention these examples merely to emphasize the discrepancies and the lack of well-established data which prevail in this field. You will notice that neither podophyllotoxin nor lapachonone is an alkaloid.
>
> "The chemical examination of wood components is rather a lengthy process. It is necessary to extract the wood with one or more solvents, *e.g.*, water, alcohol, ether, fractionate the extracts further by some suitable method, and examine the various fractions for their physiological effects. If they produce positive effects they must be further purified, for they are generally complex mixtures, and characterized chemically. I do not think there is any effective short cut to this kind of investigation."

Schwartz, Tulipan, and Peck (1947) state:

> "Wood from foreign localities or woods not handled before are the ones that usually cause dermatitis".

The workers become affected several days after beginning to work with the new wood. It is usually a sensitization dermatitis, only a few of the workers being affected. The majority of those affected become "hardened" and develop a hyposensitization after they recover. A small percentage of those affected remain sensitized.

In spite of the fact that the wood was excellent for the purpose and economical in use, the decision to withdraw the material was made because the risk of the loss of skilled personnel due to absence from work and also from those who feared contact was too great. Fine sanding is a skilled operation and workers must have long experience. The suggestion that " hardening " against the offending material is possible was also considered, but our experience with chemicals is such that increased sensitization oftens takes place and that the hyposensitization after recovery, as mentioned by Schwartz and others, appears to be rare amongst those who react strongly to exogenous irritants.

Since the first outbreak in 1953 other woods have been in daily use. In view of our previous experience over many years, particularly with hard woods of tropical origin, none of which have been troublesome, it was felt that further patch tests with these woods were not as effective as the continual handling and exposure to the fine dust, deposited on the skin and other areas which have been affected by Mansonia dust. Ordinary washing facilities have proved adequate and barrier creams have been used as a routine in the department for many years.

Clinically, the interest is directed towards the complaint of the patient that he " caught a cold in the nose ", as the irritant dust may rise and affect the nasal mucous membrane which becomes oedematous. This was noted in at least three cases where the hands of these patients were of the hyperkeratotic type and would almost certainly have offered a greater resistance to the offending dust and, as a result, the pruritus of the hands appears to have been delayed for a few days.

Summary

The outbreak of toxic symptoms from the use of Mansonia wood (*Sterculiacea altissima*) is described in which seven out of 12 men engaged on fine sanding were involved. Other cases have been known in the industry. The commonest symptoms were rhinitis, dermatitis, and oedema. It had been previously reported that collapse may occur but was not the case in any of the workers under observation. Information as to the irritant responsible is not available.

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A SURVEY OF DOCK LABOUR ACCIDENTS IN THE PORT OF LONDON

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At the close of 1952 the writer sought permission to undertake an analysis of accidents occurring in the port transport industry in London. The response from some quarters was not encouraging ; but the Port of London Authority and the National Dock Labour Board agreed to cooperate in the work, and offered full facilities. Examination of the Dock Labour Board's records revealed that these contained insufficient data for the analysis contemplated, and it was decided therefore to base the investigation on the Port of London Authority's records, supplemented where necessary by those of the Dock Labour Board. It must be pointed out that while the Authority undertakes a considerable proportion of ship discharging, transit shed, quay, and warehouse work, it does not undertake the loading of ocean vessels. To this extent the survey necessarily presents an incomplete picture of port transport operations as a whole.

The records chosen for analysis were those relating to all accidents to employees of the Authority during the year April 1, 1952, to March 31, 1953. During that time the Authority employed some 2,000 permanent or weekly labourers and an approximately equal labour force, whose composition naturally changed from day to day, of allocated or daily workers. The total of wages paid in the year to allocated workers was 3·3% greater than that paid to permanent labourers. There were, on the London Register in July, 1952, 20,694 daily workers and 9,356 weekly workers. The numbers employed by the Authority therefore are not proportional to the composition of the Port Register, and for this reason, among others, in much of what follows results for the two groups are given separately.

Collection of Information

Before analysis of the Authority's records could be begun it was necessary to list and code the various types of equipment used in port transport operations, and also those operations themselves. After some weeks of observation at the docks, and with the assistance of experienced traffic officers, a code was finally decided which was felt to be exhaustive so far as the work of allocated and permanent labourers was concerned. Supplementary codes for the cargo handled and the nature of the object actually inflicting the injury in the accident were also worked out.

The main source of information for the analysis was the Authority's standard accident report form. These reports are filed under serial numbers at the head office and it proved a comparatively simple matter, for the year under study, to extract those for all accidents to the Authority's employees throughout the Port. With the form were filed any relevant correspondence, reports of witnesses, first-aid men, ambulance room attendants and, for accidents to permanent employees involving over three days' absence, the report of an examination by the Authority's medical officer. From the information thus available a code slip was completed for every accident case, and the data thereafter transposed to Hollerith punch cards. It will be readily understood that the choice of one or other item from the codes to describe a particular accident could involve a certain amount of arbitrary judgment. To preserve uniformity, however, the writer himself completed all the more difficult items in the coding, attempting to apply a standard interpretation, and delegated only the straightforward transcription of date, time, length of absence, and worker's name, number, grade, age, and length of service. Where more than one process or piece of equipment might be said to be involved that which was proximate to the accident was chosen. For instance, if it was reported that a hand-hook slipped from a case which was being loaded on to a truck and the case fell, causing an injury, the hand-hook would be chosen as the equipment involved. Where a case was reported simply as falling from a truck, the truck was coded

as the equipment. Equipment was coded as involved in the accident if it was being operated in the process in which the accident occurred, whether mishandled or not, and whether or not the use of the equipment could be said to have caused the accident.

In practice, it was found that few accidents only were in fact difficult to code. The great majority fitted easily into one or other of the simple categories. Where items were found to be missing from the accident report file, the information was sought from other records. The length of absence for all allocated labourers in cases over three days was obtained from London Dock Labour Board records. In the treatment of results, use is not made of the average length of absence as a measure of severity rate. Where numbers are comparatively few, an undue preponderance is given in the average to absences of exceptional length, and the resulting figure can be very misleading. A better statistic is that developed by Whitfield (1944). It is the median length of absence in a logarithmic curve fitted to the observed number of cases of various lengths of absence in the group of population considered. (This median absence may be interpreted as the length of time after which 50% of the group considered will have returned from absence to work.) Where " median absence " is mentioned, it is this statistic that is referred to. Even here, however, caution is necessary in making comparisons. A preponderance of short trivial absences will reduce the median, and this therefore must only be considered in relation to the total number of cases. In every instance where a median is given it is calculated from the number of cases with absence over three days.

Results of the Analyses

Listing the cards punched from the coded reports gave for the year under review a total of 905 reported accidents for permanent labourers: 1,004 for allocated labourers, and 896 for other grades in the employ of the Authority. As a final check against the accuracy of the record keeping, coding, and punching, totals were asked for from the records kept in the actual controls at the docks and warehouses. There was a very close correspondence with a figure of 1,922 accidents for labourers as against 1,909 returned in the Hollerith list. It was assumed, therefore, that the data were sufficiently complete for the various analyses set out below.

TABLE 1
LOCATION OF ACCIDENT ON SHIP AND ASHORE

Location	Number of Recorded Accidents		
	Permanent Labourers	Allocated Labourers	Total
Hold of ship	113	165	278
Between decks of ship	7	12	19
Deck of ship	20	16	36
In craft, *i.e.*, on board barge, lighter, or elevator	101	94	195
Wharf, quayside, or lockside	115	154	269
Warehouse, shed, or vault	417	412	829
Open road	43	27	70
Railway or in rail truck	49	90	139
Open storage site	25	28	53
Other	15	6	21
Total	905	1,004	1,909

Location of the Accident on Ship and Ashore.—No adequate departmental figures were obtained for the hours worked aboard ship, in craft, or ashore; but from the point of view of accident prevention, the raw figures may still be useful. Table 1 gives the distribution and shows that by far the larger proportion of reported accidents happen within the warehouse, shed, or vault. The numbers of accidents at each point are comparatively evenly divided between permanent and allocated men save that in ship and craft work there is an apparent preponderance of accidents to allocated men in the ship's hold and to permanent men in craft. This may be due to the tendency reported to the writer

TABLE 2
SEASONAL VARIATION IN ACCIDENTS

Month	Permanent and Allocated Men			Permanent Men		Allocated Men			
	All Recorded Accidents	Cases with Absence of More than 3 Days	Cases with Absence Up to 3 Days	All Recorded Accidents	Index of Monthly Wages	All Recorded Accidents	Index of Monthly Wages	No. of Accidents Weighted for Constant Wages Index of 100	
								Cases with Absence	All Cases
January	164	45	56	66	103	98	108·6	29	90
February	171	48	63	88	90·8	83	96·1	32	86
March	183	41	60	94	105·5	89	105·1	29	85
April	108	36	45	49	94·2	59	111·3	23	53
May	158	46	59	79	107·1	79	111·3	27	71
June	169	35	48	76	97·7	93	107·7	24	86
July	189	45	52	84	102·2	105	109·9	21	96
August	146	30	36	59	94·7	87	99	19	88
September	144	29	41	72	98·2	72	93·6	24	77
October	168	35	48	87	104·3	61	89	24	69
November	160	35	53	75	102·6	85	103	28	83
December	169	41	45	76	98·5	93	106·3	17	87

TABLE 3

NUMBER OF RECORDED ACCIDENTS FOR EACH DAY OF THE WEEK

Day	Permanent Men		Allocated Men		Total	
	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence
Monday	171	60	203	58	374	118
Tuesday	178	53	175	60	353	113
Wednesday	145	39	196	61	341	100
Thursday	148	50	163	41	311	91
Friday	160	57	167	54	327	111
Saturday	72	29	59	23	131	52
Sunday	28	10	40	9	68	19
Not stated	3	—	1	—	4	—

for permanent men to fill the key positions of hatchwayman, winch or crane driver on deck, and to obtain the greater share of work in craft.

Seasonal Variation in Accidents.—Table 2 shows the reported monthly accident rate for allocated and permanent labourers combined. Again no departmental figures for the total hours worked per month were obtained and the monthly total of wages paid has therefore been taken as a measure of the population at risk. The Table also shows the monthly accident rate of permanent and allocated labourers separately and related to this is the monthly total of wages paid to each. The number of accidents to allocated men varies more closely with the total of wages than does that of the permanent men. This is understandable as the allocated man is hired only when there is work available and, in addition, the departmental figure of wages paid is not in his case biased by the inclusion of paid holidays. For this reason, the figure of accidents to allocated men is probably the better guide to seasonal fluctuations. The final two columns of the Table show the allocated accident rate weighted for a constant monthly total of wages. This weighting eliminates the need to take into consideration public holidays and variations in the number of working days in the month.

A cycle in the total of reported accidents is then apparent with peaks in midsummer and midwinter. The summer peak is not evident, however, for accidents followed by absence, although there appears to be a slight increase in winter in this category. A winter excess is the pattern usually found in industry. The sharp decline in absence in December may be noted and can probably be explained by the pressure for an undiminished wage packet before Christmas. For the comparative absence of trivial accidents (or the reporting of such accidents) in April there seems no obvious explanation. Over all, there appears little evidence to support the view that any large proportion of accidents can be attributed to working conditions in the docks involving exposure to the elements.

The Accident Rate and the Day of the Week.—There appears to be no marked fluctuation in the number of reported accidents with the day of the week. Table 3 gives the position. A slight excess at the beginning of the week with a minimum in the middle of the week is the usual finding in industry (Sargant Florence, 1916) ; Thursday is pay day for allocated men.

The Hourly Accident Rate.—The accident rate during particular hours for both permanent and allocated labourers is given in Table 4. No allowance has been made for the official tea break of from 10 to 15 minutes which is taken by the labouring grades between 9 and 10 o'clock in the morning and 2 and 3 o'clock in the afternoon. The fact that the accident gradient falls away between 4 and 5 o'clock in the afternoon is possibly accounted for by a tendency (at the time covered by the survey) to finish early, and by the last portion of the day being devoted to replacing gear, cleaning up, etc. No control figures for the number of hours worked overtime are available. The accident gradient revealed by Table 4 is seen to be steep, though equivalent figures for similar occupations are not readily obtainable as a basis of comparison. A

TABLE 4

THE HOURLY ACCIDENT RATE

Hour of Day	Number of Recorded Accidents													
	7 a.m.	8	9	10	11	12	1 p.m.	2	3	4	5	6	7	8
Permanent men	5	61	91	138	145	12	65	99	115	98	32	28	6	
Allocated men	3	59	88	136	185	13	69	104	127	109	50	40	3	
Total	8	120	179	274	330	25	134	203	242	207	82	68	9	

large amount of evidence generally, however, relating to the hourly incidence of accidents during the course of the work spell has been collected and much of it recorded in detail by a committee of the British Association (Sargant Florence, 1916). Nearly all records reveal a rapid increase during the morning to a maximum in the last hour or the last hour but one. The afternoon usually shows the same shape of increase with a lower maximum, reached earlier and falling away towards the end of the spell. The hourly accident gradient has generally been at tributed to the onset of fatigue ; but there can be little doubt that variations in the speed of production throughout the work-spell are also an important factor. Vernon (1936) writes :

> " The frequency with which accidents are incurred in the course of an industrial occupation is bound to be influenced by the speed with which the necessary movements are performed. . . . Supposing that the speed with which the repetition work is performed is increased for any reason, then, if other conditions are kept constant, the number of accidents incurred is bound to increase likewise. It is difficult to obtain reliable proof of the correctness of this statement, for any improvements of industrial output are generally due, at least in part, to improvements in the conditions of work as well."

Referring to a study of accidents to male lathe workers in a fuse factory, he says :

> " On comparing the accident curve with the output curve it will be seen that there is a considerable qualitative resemblance, though not a quantitative one. The output rose only 9% during the morning spell and 5 to 7% during the afternoon spell, so it did not show a tenth of the variations of accident frequency. However, it is probable that a small increase in hourly output may lead to a large increase in accidents. . . . Not much quickening of the actual cutting operations is permissible, so a given speeding up of output means a relatively much greater speeding up of the movements made at the times when the workers are specially liable to knock their hands against the cutting tools."

How far this argument could serve to explain the hourly accident gradient in port transport processes is doubtful. It seems at least probable that in dock labour the element of muscular fatigue would play a larger part than in lathe work. Ergographic studies summarized by Maier (1946) show the importance of allowing a sufficient refractory period in muscular effort. In repeated acts of physical exertion the time for complete recovery (that is, until the previous performance can be duplicated) increases rapidly as the period of work is increased. For example, the recovery time for 60 muscular contractions may be as long as two hours, whereas the recovery time for 30 contractions may be 30 minutes.

> " A problem in the economical expenditure of energy lies in the fact that it is unnatural for a man to rest before he is fatigued to an uncomfortable degree. Since recovery time increases rapidly as fatigue progresses, it is advisable for a workman to rest before fatigue has progressed very far or to work at such a pace that rest periods are unnecessary. The potential application of this principle can be made very dramatically by giving a man the job which required him to lift a six kilogram weight in the ergograph . . . under three different conditions. . . . Under the first condition he must make a contraction every two seconds and continues work until he is unable to lift the weight. He is then allowed to rest until fully recovered. By this procedure, the man can make 30 contractions and then must rest two hours in order to recover. In eight hours he would be able to make a total of 120 contractions. Under the second condition the man must rest after 15 contractions—from which only half an hour is necessary for recovery. In eight hours he could make a total of 240 contractions. Under the third condition the man is asked to pause ten seconds between each contraction. This pace is sufficiently slow for fatigue not to become apparent, and if extended for eight hours would accomplish 2,400 contractions (Maier, 1946)."

No such difference as this, of course, should be expected under practical working conditions, but it has been reported to the writer that experienced gangs in the docks, under good conditions and with the inducement of favourable piece-work rates, have been known to shift manually remarkably large dead-weight totals of cargo. Inexperienced attempts at the same goal with uneconomic spacing of effort might well lead to accumulating muscular fatigue not unconnected with the steep hourly gradient in accidents such as that revealed in Table 4. Only further research could help to elucidate the relative importance of speed of work and muscular fatigue as a cause of accidents in these dock labour operations. Such research might conceivably result, however, not only in a reduced number of accidents but also in actually increased production.

Artificial Lighting and Dock Accidents.—The hourly rate of accidents may also be used as a measure of the adequacy of artificial lighting. This matter was the subject of a lengthy enquiry by a Departmental Committee of the Home Office between 1915 and 1922. The findings are summarized by Vernon (1936). It was assumed that artificial lighting was in force from half an hour before sunset till half an hour after sunrise, and the accident rate during daylight and artificial light was computed from about 161,000 accidents on this assumption. An excess of some 25% for the hours of artificial light was found on a comparison of the accident frequency in the winter months, when some of the early morning and late afternoon work is done by artificial light, with that observed in the summer months. The most affected were dock

TABLE 5
ARTIFICIAL LIGHTING AND DOCK ACCIDENTS

Hour of Day	Number of Recorded Accidents (Permanent and Allocated Men)														Total
	7 a.m.	8	9	10	11	12	1 p.m.	2	3	4	5	6	7	8	
May to August inclusive	3	44	61	99	108	14	39	72	90	68	25	26	7		656
November to February inclusive	4	46	58	100	125	2	60	66	69	73	29	19	2		653

workers, with 51% excess for all types of accident and 102% excess for accidents due to persons falling. A sharp seasonal variation was found for this latter category coinciding closely with the variation in artificial lighting. Table 5 indicates the position today so far as it relates to the Authority's employees and the Port of London. Reported accidents per hour for the months of May, June, July, and August are compared with those for the months of November, December, January, and February. No increase whatever is apparent for the hours of artificial lighting in winter. These results compare favourably with those from the earlier investigation. If the figures are representative of the industry, they are a measure of the improvement from that earlier day to this.

Age and Accident Rate.—In most industrial occupations that have been studied, the accident rate diminishes with increasing age and experience, new entrants in general being particularly responsible for high rates. An attempt has been made by Newbold (1926) to separate the two factors of age and experience in a group of factory workers, with the result that with age constant the association between length of service and accidents practically vanished, whereas with length of service constant the association between age and accidents remained almost undiminished. This, however, may have been due to the small number of new entrants in the groups studied. A similar examination of the present data has not been possible. Length of service with the Authority is not commensurate with experience, as the permanent labourer, if over 21 years when taken on, invariably comes from the pool, and to have obtained the date of entry into the industry for all the cases studied was not possible under the circumstances of this survey. However, entry to the Port Register as a labourer is restricted to those over the age of 21, and the large majority enter at that age. The writer is assured that for practical purposes, at any rate for post-war entrants to the Register, age is a close indication of experience.

As stated above, in most occupations studied the accident rate diminishes with age, though the duration of absence may increase. An exception was found by Vernon, Bedford, and Warner (1928) in coal-mining where both the frequency and severity increased rapidly after the late 30s. They concluded that probably this was " due to the arduousness of the coal-miners' work. Accidents tend to diminish with age because the older men are usually more skilful and more careful than the younger men ; but on the other hand they are more easily fatigued, and fatigue may lead to accidents."

The distribution of accidents with age for the cases studied in this survey is given in Table 6. The actual age distribution of the allocated men is not available since the composition of the group, as mentioned earlier, changed from day to day. If, however, the assumption is made that the numbers employed were proportional to the wages paid, and it is further assumed that the group of allocated men employed by the Authority was a true sample of those on the Port of London Register at the time, then the numbers of allocated men in the various age groups would be those estimated in the second line of Table 6. (The calculation is only an approximation and must be treated as such. It is, for

TABLE 6
AGE AND ACCIDENT RATE

Age in Years		21–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70+
Number in Age Group (October, 1952)	Permanent	85	170	261	236	321	263	138	178	312	—	—
	Allocated (estimate)	113	217	233	244	260	226	217	165	165	121	66
Accidents with absence	Permanent	19	41	44	44	46	38	17	22	28	—	—
	Allocated	21	38	24	21	35	29	40	28	43	24	3
All reported accidents	Permanent	64	124	144	144	145	100	41	60	83	—	—
	Allocated	69	118	97	107	102	104	112	115	110	56	11
Cases (%)	Permanent	76	73	55	61	45	38	30	34	27	—	—
	Allocated	61	54	42	44	39	46	52	70	67	—	—

instance, possible that the younger allocated men on the Register, by reason of higher earnings in ship work, are attracted to employment with the stevedoring firms, and this would reflect in the age distribution of men employed by the Authority.)

If the assumptions mentioned above are accepted, however, a marked difference is evident between the two groups of men. The accident rate of the allocated falls with, but is lesser than, that of the permanent men up to the age of 45. Thereafter it rises steeply in contrast to that of the permanent group which continues to fall away. To account for this difference between the two groups, a double selective factor is probably operating. By and large, the permanent worker is picked for his reliability and physical fitness. He undergoes a medical examination before being taken on by the Authority. Those still on the daily Register after their mid-40s are likely to contain a large proportion who would be rejected for permanent employment. They are obliged, moreover, when work is allocated, to take their chance with the gang. They have less opportunity than the old among the permanent men to command the key positions such as hatchwayman, winch or crane driver, which entail less exposure to fatigue and risk of accident.

Calculation of the severity rate of absence for all cases exceeding three days serves to confirm the difference between these two groups. The calculated median length of absence (see earlier) for allocated workers under 45 is 19·5 days ; that for permanent men under 45 is virtually the same at 20 days. The median absence for allocated men between 45 and 65, however, is 30·4 days ; that for permanent men 15·5 days. The difference in the later age groups, therefore, accounts for the overall difference of 25·25 days as the median absence for allocated compared with 19·5 days for permanent men. It should be pointed out here that the permanent labourer, after six months' service, becomes entitled to benefits under the Authority's unestablished staff sick-pay scheme. Entitlement varies with length of service from one week to three months on full pay. The effect of this scheme, in making it possible for the permanent man to take absence with a measure of payment during incapacity, perhaps partially accounts for the difference between the younger groups, but would, if anything, tend to lessen the difference noted between the two older groups.

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By comparison with the cases receiving first aid those which received no immediate attention at the time of the accident appear to be followed by a disproportionate number of absences of three days or more. The greater number of sprains and strains, *i.e.*, injuries less likely to call for immediate attention, among the latter group may help to explain but cannot account for the disproportion. The Table serves to emphasize the importance of insisting on first-aid treatment, even for trivial injuries.

The distribution of all accidents by nature of injury is shown in Table 7b. The greater number of allocated cases with " injury not disclosed " is accounted for by the absence of follow-up reports on the file. Apart from this, the close approximation of the numbers of permanent and allocated men in each category supports the consistency of the diagnosis and coding in the same way that a " split-half " test would do.

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TABLE 7a
NATURE OF INJURY (TREATMENT)

Immediate Treatment	Permanent Men : No. of Cases	Allocated Men : No. of Cases	All Cases	Permanent and Allocated Cases with Absence over 3 Days			
				Total	Median in Days	Bruises, Lacerations, Punctures	Sprains, Strains
None	213	233	446	126	20	62	42
First aid at medical centre or ambulance room	472	551	1,023	107	24·9	68	24
Home and own doctor	1	2	3	—	—	—	—
Hospital	208	210	418	—	—	—	—
Unclassified	11	8	19	—	—	—	—

TABLE 7b
NATURE OF INJURY (CLASSIFICATION)

Classification	Permanent Men		Allocated Men		Total	
	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence
Bruise and contusion	334	101	375	99	709	200
Concussion	7	6	4	4	11	10
Laceration and puncture	350	64	367	64	717	128
Foreign body in eye	5	0	8	1	13	1
Rupture	2	2	4	4	6	6
Sprain, strain, dislocation	104	67	106	57	210	124
Traumatic amputation	1	1	3	3	4	4
Fracture	23	22	27	25	50	47
Burns	1	0	2	0	3	0
Shock	2	1	4	4	6	5
Industrial disease	2	2	4	2	6	4
Insect bite	2	1	2	2	4	3
Immersion	3	3	3	2	6	5
Blistering, irritation	4	1	5	3	9	4
Multiple injuries	16	7	18	12	34	19
Fatal injuries	1	—	—	—	1	—
Unclassified	48	20	72	25	120	45

operations, a further analysis was made of injuries to the foot. Of the total of 1,909 for all accidents, 222 involved foot injuries, of which 81 occurred during trucking and 37 while striking from road or rail or breaking out from pile on land. Sixty-eight cases extended beyond the third day of absence with a total of 416 days. The number and severity of foot injuries do not appear excessive in relation to the whole.

The Object Inflicting the Injury.—In the coding, provision was made for listing the nature of the object actually inflicting the injury. Though the interpretation of the word " inflicting " was necessarily arbitrary in some cases, the ratio of the numbers in each category of permanent and allocated men, whose total accidents, it will be remembered, were 905 and 1,004 respectively, again suggests that the coding was reasonably consistent. Table 8 gives the analysis. " Surface of floor and roadway " was coded here if it was alleged and not disproved to be a contributing factor in the accident. It appears to be a more popular cause for recording accidents with allocated than with permanent men, possibly because, if proved, it founds a good claim in damages. Under the heading of " part of equipment in use " were coded, amongst others, those trucking accidents in which some part of the truck struck the injured man. The excess of trucking accidents to allocated men (see later) no doubt helps to account for this similar excess here, also

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None, as in strain, rupture, or fall on good surface	90	56	101	55	191	111
Unclassified	4	2	6	2	10	4
Miscellaneous	61	19	68	17	129	36
Object on to which injured man fell, slipped, or stumbled	49	23	54	17	103	40
,, falling from above or slipping from grasp	49	18	57	20	106	38
,, ,, ,, pile or stack	66	22	77	22	143	44
,, ,, ,, vehicle or truck	47	15	54	16	101	31
,, ,, ,, moving set	17	9	20	10	37	19
,, spilling from landed set	16	5	10	1	26	6
,, jamming part of body while being moved manually	89	23	86	23	175	46
Moving set	21	6	12	6	33	12
Swinging chain, hook, grab, sling, strop	27	11	21	7	48	18
Crane hook, chain, grab, etc., handled with crane	13	6	12	2	25	8
Tautening or running wires, chains, or ropes	2	0	3	1	5	1
All other wires, chains, ropes	3	1	1	0	4	1
Moving road vehicle	7	5	6	2	13	7
Moving power truck	6	4	5	1	11	5
Part of equipment in use	89	20	123	43	212	63
Tool in use	77	14	71	13	148	27
Projecting sharp edge, splinter, or nail	56	9	61	8	117	17
Metal or wire binding	50	7	65	8	115	15
Movable object on floor, deck, or ground	39	13	36	9	75	22
Fixed object on floor, deck, or ground	6	5	2	1	8	6
Grit	6	0	7	2	13	2
Surface of floor or roadway	11	5	41	20	52	25
Noxious material	4	1	5	1	9	2

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Fracture	23	22	27	25	50	47
Burns	1	0	2	0	3	0
Shock	2	1	4	4	6	5
Industrial disease	2	2	4	2	6	4
Insect bite	2	1	2	2	4	3
Immersion	3	3	3	2	6	5
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Unclassified	4	2	6	2	10	4
Miscellaneous	61	19	68	17	129	36
Object on to which injured man fell, slipped, or stumbled	49	23	54	17	103	40
,, falling from above or slipping from grasp	49	18	57	20	106	38
,, ,, ,, pile or stack	66	22	77	22	143	44
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,, ,, ,, moving set	17	9	20	10	37	19
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,, jamming part of body while being moved manually	89	23	86	23	175	46
Moving set	21	6	12	6	33	12
Swinging chain, hook, grab, sling, strop	27	11	21	7	48	18
Crane hook, chain, grab, etc., handled with crane	13	6	12	2	25	8
Tautening or running wires, chains, or ropes	2	0	3	1	5	1
All other wires, chains, ropes	3	1	1	0	4	1
Moving road vehicle	7	5	6	2	13	7
Moving power truck	6	4	5	1	11	5
Part of equipment in use	89	20	123	43	212	63
Tool in use	77	14	71	13	148	27
Projecting sharp edge, splinter, or nail	56	9	61	8	117	17
Metal or wire binding	50	7	65	8	115	15
Movable object on floor, deck, or ground	39	13	36	9	75	22
Fixed object on floor, deck, or ground	6	5	2	1	8	6
Grit	6	0	7	2	13	2
Surface of floor or roadway	11	5	41	20	52	25
Noxious material	4	1	5	1	9	2

TABLE 9
CARGO HANDLED AT TIME OF ACCIDENT

Classification	Permanent Men		Allocated Men		Total	
	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence
None	194	72	189	66	383	138
Cargo not stated	31	13	37	11	68	24
Small unpacked cargo	7	5	10	3	17	8
Cases	178	56	239	69	417	125
Crates	16	4	13	4	29	8
Cartons	23	5	31	4	54	9
Tins and metal containers	2	1	6	1	8	2
Barrels, casks, kegs	51	11	18	3	69	14
Hogsheads	9	4	2	1	11	5
Drums	18	9	23	9	41	18
Glass and bottles	6	2	2	1	8	3
Bags	96	33	97	34	193	67
Bales (other than rubber)	60	17	37	10	97	27
Baled rubber	15	4	18	5	33	9
Bulk grain and seeds	4	0	30	7	34	7
Bulk sugar	7	5	3	2	10	7
Carcases	29	6	34	8	63	14
Hides, skins	6	1	0	0	6	1
Ingots	14	5	8	3	22	8
Rails, pipes, tubes	15	4	27	6	42	10
Uncased vehicles or machinery	2	1	4	3	6	4
Sawn timber	30	15	66	30	96	45
Logs	3	2	14	7	17	9
Floated logs	1	1	1	0	2	1
Plywood	21	8	15	4	36	12
Stone, marble	15	4	8	2	23	6
All not listed above	52	10	72	14	124	24

that under the heading of " Object falling from vehicle or truck ". Other items are self-explanatory and need no comment.

Cargo Handled at Time of Accident.—Cargo was coded with particular reference to the type of packaging. No control figures were obtained for the total weight of any category handled and the risk of accident associated with each cannot therefore be calculated. The relative overall effect of any steps in accident prevention that might be taken in relation to a particular type of cargo can be judged, however, from the comparisons in Table 9. It may be of interest to note that of the 446 accidents occurring when cases and crates were handled 179 were associated with the operation of two-wheeled and four-wheeled hand-trucks, 33 with the use of hand- and pad-hooks, while 190 were not associated with the use of any equipment. Of the 290 accidents occurring when bags and bales were handled, 86 were associated with the use of hand- and pad-hooks, 28 with the quay-crane, and 121 were not associated with the use of any equipment. No other type of equipment was predominantly associated with accidents involving these two categories of cargo.

Equipment Involved in the Accident.—As explained earlier, equipment was coded as being involved if it was being operated at the time of the accident and in the process in which the accident occurred, whether that equipment was mishandled or not. This was perhaps the easiest to apply of any definition of " involvement " but it was, even so, difficult to interpret with varying individual sets of facts. The interpretation, moreover, was of course limited to the detail of the report in the accident file and to the accuracy of the statements given. With these limitations, Table 10 illustrates the analysis of equipment and accidents. One striking result is that for approximately a half of all accidents reported no equipment is coded as being involved. Another is that the hand-truck is by far the biggest contributor numerically to the total of accidents. With the hand-truck also it will be noted that the accidents to allocated men are over twice as numerous as those to permanent men. This is explained to the writer as the result of the majority of allocated labour being employed hand-trucking in ship's gangs. With the mobile crane manned, by contrast, predominantly by permanent gangs, the majority of accidents would be expected to occur to permanent men. The category of accidents with no equipment involved provides again an interesting comparison between allocated and permanent men. Here we have two roughly equal groups, with an almost identical number of recorded accidents of this type. The median absence for permanent men is 16·5 days, for allocated men 24·25 days. The median for the combined total is 20·5 days.

The median length of absence for the two-wheeled hand-truck, 26·75 days, shows that the accidents that occur with this equipment besides being the highest numerically are by no means trivial in severity. The same median holds for the

TABLE 10
EQUIPMENT INVOLVED

Classification	Permanent Men		Allocated Men		Total	
	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence
None	484	170	485	160	969	330
Unclassified	6	0	9	0	15	0
Quay crane	59	25	46	16	105	41
Mobile crane	30	14	9	4	39	18
Cranes (other than above)	8	3	1	1	9	4
Capstan	1	0	0	0	1	0
Winch	3	1	7	4	10	5
Ship's derrick	3	0	3	2	6	2
Conveyor	2	0	1	0	3	0
Road vehicle	3	0	2	0	5	0
Rail vehicle	3	0	8	0	11	0
Fork-lift truck	1	0	2	1	3	1
Electric truck	32	13	35	9	67	22
Tractor and trailer	12	0	6	0	18	0
Grain elevator	2	0	21	7	23	7
Collis and similar truck	15	6	46	19	61	25
Hand-truck (2 wheels)	83	24	170	42	253	66
" " (4 wheels)	16	5	28	9	44	14
Barrow	1	0	1	1	2	1
Hand-hook	63	13	74	13	137	26
Pad-hook	3	3	2	1	5	4
Hand tool (other than hook)	3	2	3	1	6	3
Wires, ropes, chains, slings	8	2	4	0	12	2
Gangway plank	0	0	8	5	8	5
Toe board and staging	4	1	4	0	8	1
All not listed above	60	16	29	12	89	28

total of accidents with all types of hand-truck, *i.e.*, with doomie, collis, and four-wheeled hand-trucks included. By contrast the hand-hook, with a median absence of 9·75 days, is shown to be an unimportant contributor from the point of view of severity, although numerically it contributes nearly as many accidents to the total as all types of crane combined. The rather fearsome appearance of this tool seems to have dramatized its importance in the minds of many who have discussed with the writer the causes of dock accidents. Similarly the apparent harmlessness of the two-wheeled truck has probably led to an underestimating of its importance as a cause of injury. Accidents with capstan, winch, and ship's derrick appear to be rare (at any rate to the Authority's labourers) and again their dramatic nature, when they do happen, appears to earn them an undue importance.

The crane (all types, save for the mobile crane) is seen as the most dangerous piece of equipment in use, as was expected. The severity rate, a median absence of 38·6 days, is higher than for any other equipment. When the figures for the mobile crane are included in this total, the median drops considerably to 28·6 days. The electric truck with a median of 24·5 days of absence is not more dangerous than the hand-truck.

An interesting fact to note is the absence of accidents reported for the fork-lift truck. It is possible that this equipment by eliminating the manual handling of cargo and with protective shields for the driver, considerably reduces the accident rate. Control figures for the amount of cargo shifted and the number of individuals at risk were not, however, available.

Process in which Accident Occurred.—In the coding and analysis of the tasks which together make up the port transport processes care was taken to list the largest number of separate activities that the accident reports were likely to describe. Table 11 gives the analysis. Once again attention is drawn to the trucking processes, which contribute nearly a quarter to the total of all accidents. It is also interesting to note that when the truck is being loaded there are four times as many accidents as when being unloaded.

The Authority's labourers would be engaged making up sets and slinging both ashore, in craft, and in the ship's hold. As they do not load ocean-going vessels, however, they would be landing or breaking down sets only ashore or in craft. This possibly accounts for the lesser number of accidents in the breaking down process.

Conclusions

The foregoing analyses give a fair indication of where, when, how, and to whom accidents happen amongst the Authority's labouring employees. In the absence of any contrary evidence, and with the reservation that ship loading has not been considered, there seems reason to believe that the findings would apply equally well to the labour force employed in the Port of London as a whole, and possibly even to port transport processes in general. One encouraging inference from the

TABLE 11
PROCESS IN WHICH ACCIDENT OCCURRED

Classification	Permanent Men		Allocated Men		Total	
	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence	All Recorded Accidents	Cases with Absence
Process related to work of other grade	6	3	5	3	11	6
,, ,, irrelevant to cause of accident	11	9	22	9	33	18
Unclassified	13	5	14	1	27	6
Miscellaneous	97	22	61	20	158	42
Moving to point of work over ground floor or deck	31	14	35	20	66	34
,, ,, ,, ,, ,, on steps	6	3	2	1	8	4
,, ,, ,, ,, ,, on ladder	5	2	7	5	12	7
,, ,, ,, ,, ,, over cargo	11	5	11	5	22	10
Leaving or entering vehicle or crane	12	4	7	2	19	6
Preparations (miscellaneous)	3	2	3	3	6	5
Opening or closing doors or gates	13	2	20	6	33	8
Removing and replacing hatches	8	4	15	2	23	6
Collecting or returning gear	3	0	1	0	4	0
Gearing	3	0	8	2	11	2
Shifting cargo (miscellaneous)	4	1	9	3	13	4
Striking from road	41	10	38	10	79	20
,, ,, rail	25	12	30	6	55	18
Breaking out from pile on land	48	20	38	14	86	34
,, ,, ,, ,, stow in ship or craft	36	8	61	22	97	30
Other manipulation of cargo by hand or with hand tool	35	7	28	5	63	12
Transporting cargo manually	14	6	18	7	32	13
Shovelling or trimming bulk cargoes	7	5	24	7	31	12
Making up set and slinging	83	31	83	27	166	58
,, ,, set on pallet board	2	0	0	0	2	0
Landing case or set and breaking down set	42	15	34	9	76	24
Manipulating cargo at scale	20	12	5	0	25	12
Rafting or working floated timber	1	1	1	0	2	1
Piling or stowing cargo on shore	44	9	47	7	91	16
Stowing in ship or craft	33	9	15	6	48	15
Delivery (miscellaneous)	1	1	0	0	1	1
Loading cargo on to van or lorry	20	5	17	6	37	11
Delivery to rail	4	1	5	4	9	5
Trucking (unclassified)	7	3	3	1	10	4
Loading cargo or equipment on to truck	71	17	125	33	196	50
Transporting cargo by truck	52	22	118	34	170	56
Unloading cargo from truck	21	6	31	9	52	15
Driving or pushing empty truck	15	7	23	6	38	13
Working with crane. Driving crane	7	2	0	0	7	2
Fixing crane hook, grab, or dog to set or cargo	19	4	17	5	36	9
Releasing crane hook, grab, etc.	9	2	11	4	20	6
Manning hatchway	3	0	0	0	3	0
Other operations. Marking and tallying	8	4	3	2	11	6
Opening and closing packages	11	3	9	1	20	4

analyses reported in this paper is that perhaps the most promising way to improvement in safety may not lie in the direction of imposing irksome restrictions which also act incidentally as an impediment to work. It may well be that attention to the better pacing of physical effort, and the increase of mechanization by the use of fork-lift trucks in the trucking processes, will be shown to lead to a reduction in accidents simultaneously with an increase in production. It is possible also that a redesigning of the ordinary two-wheeled hand-truck might lead to a reduction in accidents. A shield against falling packages could perhaps be fitted without impeding the handling qualities of the truck, and yet at the same time preventing it from tipping. As this simple piece of equipment, the hand-truck, is in general use throughout industry, the matter is worth attention.

Summary

A survey was carried out of all reported accidents to employees of the Port of London Authority in the year April 1, 1952, to March 31, 1953. Codes were first drawn up for the personal particulars of the worker injured, for the process involved, equipment in use, cargo handled, object inflicting the injury, and the time, date, and location of the accident. The information from each of 1,909 accident reports was transposed to punch cards and sorted mechanically.

The number of accidents to permanent and allocated labourers was roughly equal, but a greater length of absence was found amongst allocated men. This difference was found to be accounted for by the group of allocated men over 45 years old.

Small seasonal peaks in minor accidents were found in summer and winter, but no evidence to support the view that a large proportion of accidents could be attributed to environmental conditions. A steep hourly accident gradient was apparent, possibly related to an uneven pacing of work. There was no increase in accidents during the winter hours

of artificial lighting. The handling of cases and crates contributed the largest total numerically for cargo handled, but control figures of the relative amounts of cargo handled were not obtained.

Accidents in which cranes were involved were the most severe with a median absence of 38·6 days. The hand-truck, however, was by far the biggest contributor numerically to the total of accidents, and the cases in which this equipment was involved were also severe with a median absence of 26·75 days. Hand-hook accidents though numerous were trivial in severity.

The writer's grateful thanks are due to all on the Authority's staff who assisted in this research, especially those who helped with the irksome task of coding accident reports. Thanks are also due to Dr. N. H. Mackworth, with whose encouragement and under whose direction this work was undertaken.

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THE OCTOBER (1955) ISSUE

The October (1955) issue contains the following papers :—

A Clinical Study of a Group of Accident-prone Workers. By James A. Smiley.

Coal-miners' Pneumoconiosis in Four Collieries in County Durham. By R. I. McCallum and R. C. Browne.

The Energy Expended While Walking in Stooping Postures. By Thomas Bedford and C. G. Warner.

Tolerance of Men to Work in Hot, Saturated Environments with Reference to Mines Rescue Operations. By A. R. Lind, R. F. Hellon, J. S. Weiner, and R. M. Jones.

The Formation of Phosgene by the Action of Hot Surfaces and its Absence when Tobacco is Smoked in Atmospheres Containing Chlorinated Hydrocarbon Vapours. By J. Little.

Older People and Heavy Work. By R. M. Belbin.

Dermatitis from Solutions of Sodium and Calcium Chloride in the Cola-mining Industry. By O. P. Edmonds.

Miscellanea :

Four Studies of the Relation between Psychological and Occupational Handicap among Industrial Workers. By Alastair Heron.

A Method for the Determination of Carbon Monoxide in Blood. By P. J. Lawther and G. H. Apthorp.

British Occupational Hygiene Society : The Assessment of Health and Disease in Industry.

The Diseases of Occupations. A review by Richard Schilling.

Book Reviews.

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MISCELLANEA

A Study of Variations in Output

I. M. RICHARDSON and W. Z. BILLEWICZ*

From the Departments of Social Medicine and Midwifery, University of Aberdeen

The importance to industry of variations in working efficiency and their causes has been recognized for many years. Between the wars a considerable number of investigations was sponsored by the Industrial Health Research Board and in recent years interest in output variations has increased. The approach of particular research workers has differed widely; the influence on output of hours of work, rest pauses, environmental conditions, monotony and variety of tasks, eyestrain, methods of work, shift systems, mental handicap, are only some of the factors studied. In most published papers, whatever their special approach, much attention is devoted to the several and severe difficulties inherent in the use of output figures as an index of human efficiency. After long experience in this field Wyatt (1944) stated that "factory output records must be interpreted with the greatest caution. Normally variations of output are rarely, if ever, due to a single outstanding cause".

Broadly speaking, variations in performance are caused by a combination of personal and impersonal factors. Efficiency may be disturbed by changes in materials, machines, methods of production, working conditions and other factors, without any apparent change in the physical or mental state of the worker; on the other hand conscious or unconscious changes in the worker can alter output in the absence of any detectable change in the material and mechanical conditions of the job. Since both sets of variables are constantly interacting it can be readily understood how complex is the aetiology of output variations.

Obviously the extent to which a worker is able to control his material and machine must be an important determinant of the amount of variation. At one extreme is the situation where the influence of mechanical factors is almost negligible, as for example in some of the handcrafts, at the other is the automatic machine which requires only starting, stopping, and a constant feed of material. In all studies of output this element of control is outstandingly important.

In 1920 Wyatt reported an investigation of output variations among cotton mule spinners in which he showed that "in mule spinning the part played by the operative is very small compared with the overwhelming influence of the machine". Any technical advances in the last 30 years have tended to reduce still further the operator's control over a spinning mule. Despite this high degree of mechanization our findings show that individual differences between workers can be an important source of output variation.

Method of Study

We were fortunate in having free access to a modern woollen mill where for five years a production-study department had been developing. The aim of this department was to establish a sound scheme of payment-by-results based on work-study techniques; after intensive study of the duties of "piecers" (the name given to operators of spinning mules), standard times per unit of material had been calculated and a formula devised which measured output in performance units per week. These operator performance figures are hereafter referred to as O.P.

Weekly O.P. figures for 36 operators (33 women and three men) were collected from production-study records which were scrutinized while "hot", *i.e.*, at the end of each week, and, with technical assistance, notes were made about any mechanical or material peculiarities. Although the O.P. formula was designed to equalize operators in respect of both these factors, and probably did so with small peculiarities, we decided to reject all O.P. figures for weeks in which a major mechanical breakdown or exceptional quality of wool had been recorded. By so discarding 26 O.P.s the amount of variation due to non-human sources has been reduced; we are satisfied that any remaining variation due to these factors is probably of minor importance and would in any case have affected all operators in the same degree.

The average period of observation per operator was 33 weeks. Absence records were kept throughout the study and for all absences of one week or longer medical evidence was available. This "health" information was supplemented by a personal interview with each worker at the end of the study; no medical examination was carried out, but subjective health (including any symptoms or disabilities) was assessed by careful questioning. One of us knew most members of the group in advance and was therefore able to establish good rapport at this health interview. None of the group was aware that we were observing their output, the interview being presented as part of a normal interest in occupational health.

It must be emphasized that our study was concerned with individual performance, not with variation in the weekly production of the spinning department as a whole. By weekly output figures are meant the performances of the 36 operators selected for study during the weeks when they were at work.

Findings

Operators were divided into two groups: (1) 29 operators in good health and (2) seven operators whose

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physical condition suggested a possible source of output variation. This group comprised a totally deaf girl, a middle-age woman with hypertension, an elderly man severely disabled by emphysema, and four pregnant women.

The Effect of Experience.—The two groups were subdivided according to length of experience in piecing. Six experience groups were distinguished: first year, second year, third year, fourth and fifth years, sixth to fourteenth year, and fifteenth year or more. We chose this grouping partly in the belief that small differences in experience are likely to be more important at the beginning of an operator's career whereas wider grouping is permissible with more experienced workers, and partly because it gave groups of roughly even size.

Table 1 shows the mean O.P. and standard deviation for each of the 29 healthy operators grouped by length of experience. The data show that average weekly performance tends to rise as experience increases. This upward trend may be due in part to a selection effect, the less efficient operators having dropped out in the first few years leaving their more skilled contemporaries to move on into the upper experience groups. If this had been happening the comparison between lengths of experience may be false but against this view is the fact that only those operators who, during training, showed promise of becoming proficient, were allowed to become production piecers; this policy "weeded out" the least efficient and must have reduced the selection effect here considered.

In the further analysis we have omitted records of the three girls with only one year of experience. In the mill it was well known that, after her training period with an experienced piecer was over and she was given a mule of her own, a girl's performance improved rapidly in the first few weeks of independent work. This improvement shows in production records not only as rising output but also as diminishing fluctuations; consequently neither the early mean nor its standard deviation is very meaningful when compared with more experienced groups. This point is illustrated in Figs. 1 and 2 where the output graph of a beginner is compared with that of the most variable operator in one of the upper experience groups.

Group (1).—This consisted of 26 operators in good health and with more than one year of experience.

If during a given week all the operator performances showed a rise or fall, the explanation would probably be some common factor such as material shortage, mechanical breakdown due to power failure, an emergency demand for high output, or perhaps the imminence of a holiday. During the period of study none of the first three situations arose nor did a close inspection of the records reveal any weeks in which the performance of all or most of the workers improved or deteriorated. All weeks can therefore be regarded as normal in this respect.

The data in Table 1 suggest that operators differ not only in average performance but also in their internal, *i.e.*, within the operator, variability. Bartlett's test for homogeneity of internal variances yields a χ^2 (25) of 126·5; this result is highly significant and shows that there are real differences between operators in respect of the size of their output fluctuations. It is interesting to observe that there seems to be no consistent relationship between average performance and its variability. Since the number of weeks of observation varies from operator to operator, it may also be stated —to clear the way for further analysis—that there does not appear to be any relation between the size of the internal variance and the length of the period of observation. One explanation for the heterogeneity of internal variances could be that some workers had long spells on the same material whereas others were more frequently switched between different yarns; this of course assumes that the O.P. formula did not make adequate allowance for such events. However, scrutiny of the records revealed no evidence that some operators were more favoured with "runs" of material than were others.

Since mule spinning is highly mechanized and because the operation of "piecing" is not a complex manual skill, one might expect that differences between individual operators would contribute little in the analysis of total variation. This point can be investigated if (while recognizing that for individuals its value may be substantially greater or smaller) the estimate of average internal variance of operators is accepted as meaningful.

TABLE 1

MEAN OPERATOR PERFORMANCE (O.P.) AND STANDARD DEVIATION IN 29 HEALTHY OPERATORS BY LENGTH OF EXPERIENCE

Piecing Experience															
1st Year (3 Operators)			2nd Year (6 Operators)			3rd Year (5 Operators)			4th and 5th Years (4 Operators)			6th–14th Years (6 Operators)			15th Year or More (5 Operators)		
Mean O.P.	S.D.	Weeks Observed	Mean O.P.	S.D.	Weeks Observed	Mean O.P.	S.D.	Weeks Observed	Mean O.P.	S.D.	Weeks Observed	Mean O.P.	S.D.	Weeks Observed	Mean O.P.	S.D.	Weeks Observed
128·7	6·6	15	139·3	3·2	39	141·9	5·4	35	140·0	7·2	36	149·0	3·3	32	142·5	2·7	20
133·7	6·2	32	139·7	2·7	41	140·4	5·2	39	144·4	6·0	35	151·8	4·6	37	145·7	5·8	24
136·0	5·2	38	138·7	4·1	41	140·0	3·6	40	143·8	5·0	36	142·3	3·6	21	149·0	4·6	39
—	—	—	135·3	7·6	37	133·0	6·3	34	141·0	4·2	40	144·6	5·5	28	148·9	6·1	40
—	—	—	136·0	3·5	34	140·2	5·3	41	—	—	—	139·3	2·9	18	140·4	3·8	41
—	—	—	136·3	4·6	33	—	—	—	—	—	—	138·3	5·1	32	—	—	—
Group Mean O.P. 133·8			*Group* Mean O.P. 137·7			*Group* Mean O.P. 139·2			*Group* Mean O.P. 142·2			*Group* Mean O.P. 145·0			*Group* Mean O.P. 145·0		

FIG. 1.—The output chart of a woman operator, single, aged 26 years, in good health and starting work as a piecer.

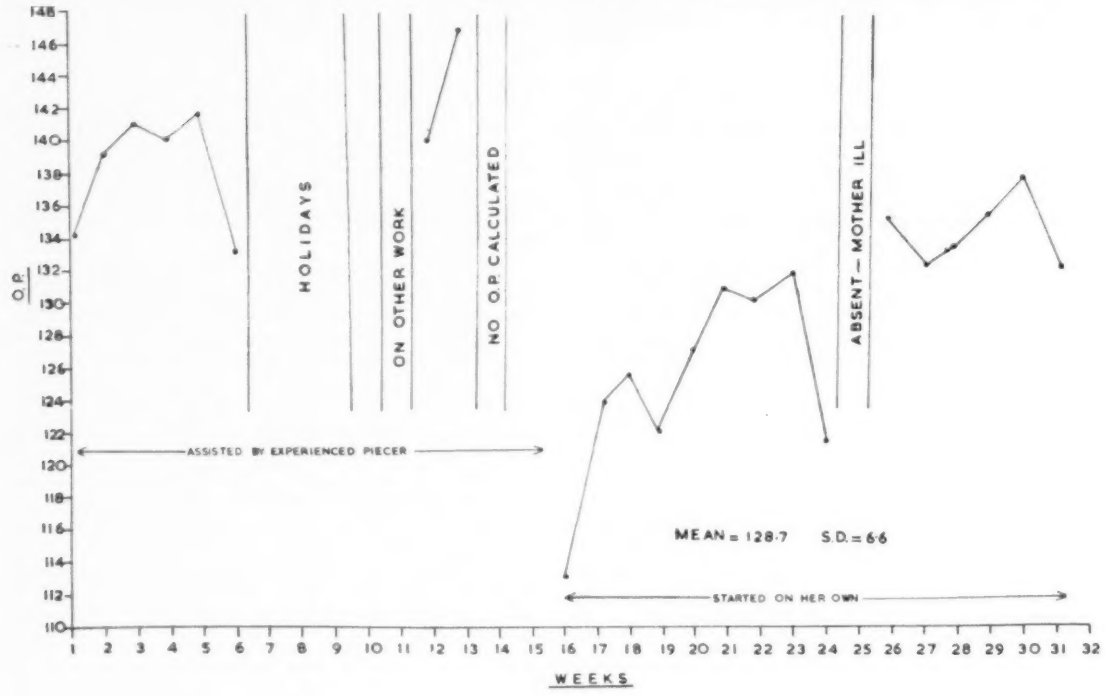
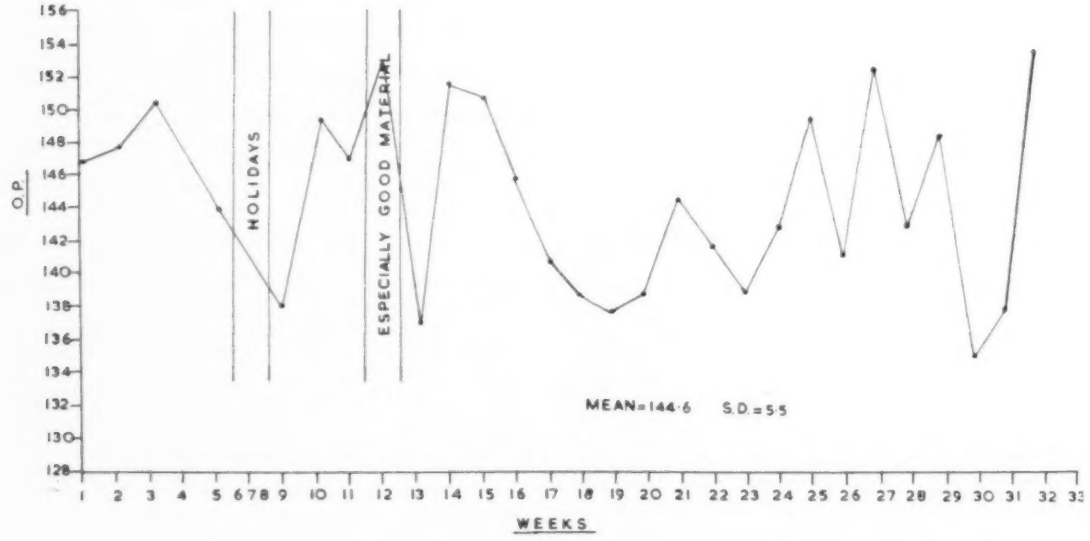


FIG. 2.—Output chart of a woman operator, married, aged 37 years, in good health except for bilateral varicose veins, with 10 years' experience.



To allow for the influence of experience, the sum of squares attributable to differences between experience groups has been removed; Table 2 shows the partitioned sum of squares and the relevant mean squares.

TABLE 2

'OPERATOR PERFORMANCE' VARIATION OF 26 HEALTHY OPERATORS

Variation	d.f.	Sums of Squared Deviations	Mean Square
Between experience groups* ..	4	9,050·28	2,262·57
Between operators within experience groups	21	9,386·61	446·98
Within operators	867	21,156·79	24·40
Total	892	39,593·68	44·39

* Experience groups, 2nd year and over.

From this Table the average internal variance of an operator $\overline{\sigma_i^2}$ is estimated by $s_i^2 = 24{\cdot}40$. Taking into account the varying number of weeks of observation (given in Table 1), there can be estimated the average component of variance, σ_b^2, attributable to differences between an operator's performance and the average performance of operators with similar experience; this component is $s_b^2 = 12{\cdot}32$. This latter component of variance is about 50% of the average internal variance of an operator and we therefore conclude that even in a highly mechanized process the differences between individual operators form a substantial part of total variation.

Since the conditions of work were uniform for the group and the 26 operators were in good health, we suggest that the foregoing differences in average output and internal variability point to real variations between operators in respect of mental capacity and/or inclination for work.

The weekly records of output revealed no sign of lower or higher performance in the periods immediately before or after sickness absence from work. This was certainly true of short absences, most of which were due to upper respiratory ailments; there were very few cases of more serious illness, e.g., pleurisy, bronchitis, which had caused absence of several weeks, but even in these records no suggestion was found of a relationship between illness and output.

Group (2).—For convenience the three operators with physical handicap and the four pregnant girls are considered together. Fig. 3 shows the scatter of average O.P.s in experience groups.

The circle in the first year experience group denotes a girl who is totally deaf since birth but, though her training presented some difficulty, her deafness did not appear a handicap in the operation of piecing and she was regarded by the supervisors as a very competent worker.

The upper circle in the most experienced group is the mean O.P. of a 49-year-old woman absent for three weeks with hypertension and still under medical supervision at the end of the study. The lower circle is the mean output of a 65-year-old man with severe emphysema and chronic bronchitis who had to retire from work on health grounds soon after the study was completed.

Only one of the four pregnant piecers shows an output below the range of her experience group. A complication of pregnancy forced her to leave before the end of the investigation.

It appears that with two exceptions the performance of operators with a potential disability was not markedly different from the performance of the healthy group.

The observation that the average performance of the four pregnant operators was not lower than that of the others is by itself not evidence that performance was unaffected by pregnancy. In Table 3 are shown the mean O.P.s of these four operators before and during pregnancy.

TABLE 3

MEAN O.P. OF FOUR OPERATORS BEFORE AND DURING PREGNANCY

Piecing experience (years) ..	3	5	5	8
No. of weeks worked in pregnancy	13	26	12	13
Before pregnancy				
Mean O.P...	136·8	140·0	139·6	136·0
Weeks observed	29	23	5	4
During pregnancy				
Mean O.P...	139·6	141·8	141·6	134·8
Weeks observed	12	21	7	13

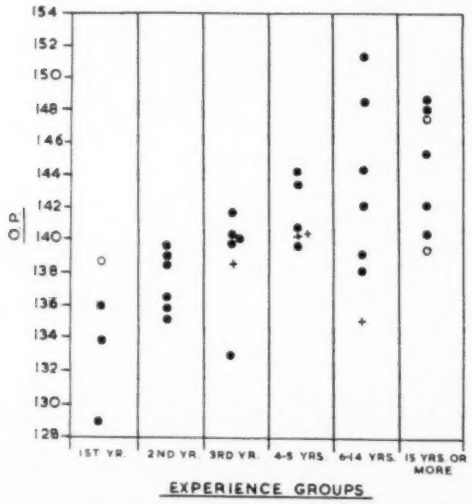


FIG. 3.—Comparison of healthy operators with selected group.
● average O.P. = healthy groups.
○ ,, ,, = impaired, healed, or disability.
\+ ,, ,, = pregnant operator.

From such slender data inference must be very tentative but it seems worthwhile to point out that, despite a good deal of pregnancy nausea and sickness, none of the operators showed any serious deterioration of output in early pregnancy.

Discussion

If output is to be used as an index of mechanical, material, environmental, or individual variations, detailed knowledge of its formulation is essential. In this study the units of output were the product of elaborate work-study techniques adopted by the firm as the best basis for an incentive scheme. For that purpose the output measurements were satisfactory. It is well known that there are dangers in using records for purposes other than those for which they were designed and we have therefore been mindful of the possibility that the O.P. formula was not sufficiently sensitive to reveal an association between health and output.

From the data presented there is no evidence of a relationship between physical health and output. We have suggested that an important source of variation of output lies in differences between operators in respect of (*a*) experience and (*b*) attitudes and aptitudes. Had we been able to assess mental health over the study period our findings might have been more closely related to what, for lack of a more precise expression, may be termed "total health." Markowe and Barber (1952, 1953) have made important contributions to the relationship between mental health and productivity. Their findings show some of the complexities of this relationship and emphasize the need for more intensive studies of the influence of social background at home and at work. During our own study the senior foreman commented several times on the possible association between output and various domestic situations known to him, but in the absence of systematic inquiry this association could not be pursued.

Two final points merit attention. We could discern no sign of conscious restriction of output; during 1954 demand for production was high, there were ample supplies of yarn for the spinning mules, and all piecework earnings were calculated on individual output. Secondly, there is the question of relationships between workers and management; in this firm there was a long and honourable tradition of employer-employee good will, evidence of which was provided by the ease and cooperation with which the work-study techniques were accepted in the spinning sheds. Throughout our period of observation, attitudes of mutual respect were so discernible that, though we have no specific data on morale, the existence of serious group discontent was unlikely. But we readily admit that, without a careful investigation similar to that of Davis (1953), appearances may have been deceptive.

The last conclusion from this preliminary study concerns the research opportunities afforded by good work-study schemes. Doctors with access to industries using these schemes are most favourably placed for the investigation of health factors in efficiency at work—provided always that the techniques and records are understood and their limitations appreciated.

Summary

The intra-individual and inter-individual differences of output have been studied in a group of 36 spinning mule operators, seven of whom had physical disabilities.

(1) From the data on those in good health it is suggested that variations in performance are likely to have been due to differences in capacity and inclination for work. The effect of experience is discussed.

(2) A group of three disabled and four pregnant workers had performances similar to the healthy group.

(3) No change of output could be discerned before or after sickness absence from work.

(4) The investigation points to the importance of studying mental health in relation to output.

We wish to thank the management of J. & J. Crombie, Ltd., in particular Mr. N. Howarth of the Spinning Department, for generous cooperation. We are much indebted to Mr. S. Wyatt for helpful criticism.

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Shell Haven Medical Centre

GUNILLA LIDDLE

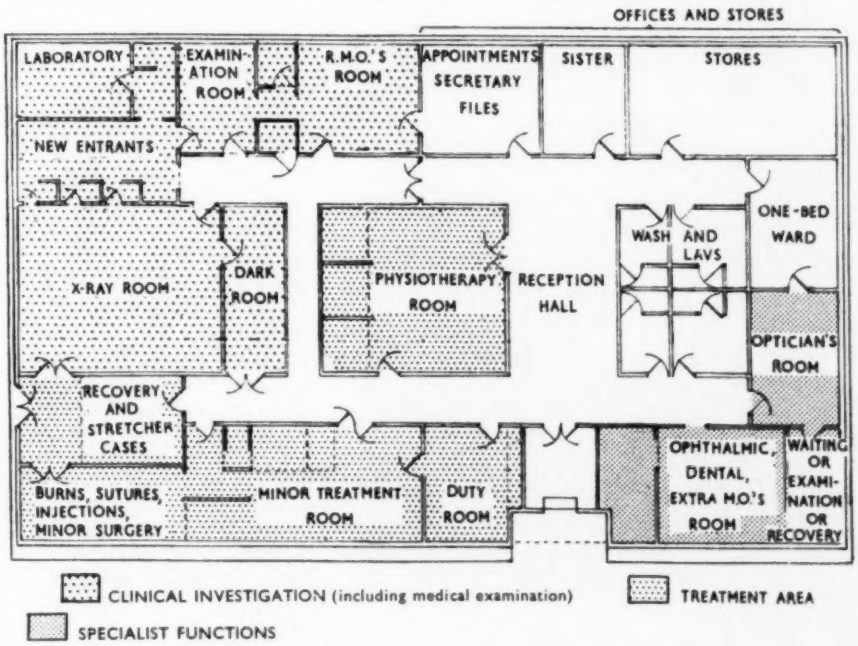
Today the concept of "occupational medicine" is that of Thackrah and other pioneers, but embracing the experience and practice gained in the industrial world of the past 100 years. The medical centre at Shell Haven, Essex, which has been built to cater for 2,000 permanent employees at the refinery and up to 2,000 contractors' employees working on extensions to the refinery, would, we venture to think, have pleased Thackrah. For the Shell Haven Medical Centre, in addition to achieving its primary purpose, is fitted to be the expression of that other aspect of occupational medicine lost sight of for a time but today taking its proper place again—research by the "ordinary" industrial medical officer. A well planned centre of bricks and mortar, an adequate staff, and several thousand operatives exposed to certain definable hazards all provide his natural experimental milieu.

The Shell Haven Medical Centre is a one-storey building, planned and built in such a way that modifications or additions can easily be made as future needs demand. The most modern devices have been employed to heat, light, ventilate, and, most important, make it sound-proof.

The first impression as one enters the Centre—and this must also affect the patient—is the exhilarating use of colour. Strong blues and yellows with one flame coloured wall confront the patient as he enters the hall and presses a button to announce his arrival. As soon as the doctor or nurse is ready to see him a light glows and he immediately goes for his treatment or interview. This procedure is in operation throughout the 24 hours. The treatment room—coloured less vividly, but not dully, in blue and cream—is divided into two units, by cupboards containing drugs and dressings, for minor treatments and for clean treatments. In the former, infected wounds are dressed, skin diseases treated, all by the "no-touch" technique. In the latter, clean wounds are sutured, injections given. A corner of the minor treatment end is curtained off so that eye injuries may be examined and treated.

Careful arrangements have been made for the reception of the seriously injured. Ambulances draw up in a covered bay leading to a quiet, darkened recovery room. Doors from the recovery room open into the treatment room on one side and into the *x*-ray room on the other. There are all the essentials for resuscitation, including a "minuteman" resuscitator. Afterwards the patient can be given an *x*-ray examination and first-aid treatment and then sent home or to hospital. The exact position of the recovery room in relation to the rest of the Centre is important, because the presence of a seriously injured man is not known to those attending for minor ailments.

There are certain well recognized hazards in an oil refinery, and a laboratory and *x*-ray unit are essential to



the preventive aspect of medicine there. At Shell Haven routine chest films are taken of all who join the company and all who go overseas and in addition of tetraethyl lead handlers, chemical product handlers, chemical plumbers, benzene workers, alkylate workers, and tank cleaners. In the laboratory full blood counts are performed at six-monthly intervals on tetraethyl lead workers, benzene workers, chemical product handlers, chemical plumbers (on whom stipple counts are also done), tank cleaners, and industrial radiographers. Routine urine examinations are also performed on all these groups of workers, and as new processes are developed the medical officer joins the consultations with the planners so that new hazards may be anticipated and, if necessary, new laboratory control be instituted in those workers exposed to new and perhaps unpredictable hazards to health.

Certain auxiliary services also find a place in the medical centre : these are physiotherapy, an ophthalmological service, and a dental service. Each specialty has its own set of rooms, and, under the N.H.S., an ophthalmologist, a dentist, and an optician visit the Centre to treat and prescribe. Simple forms of physiotherapy are carried out by the Centre's own staff, and their rooms can also be used as rest-rooms. The specialist services in particular cut down the number of wasted man-hours in which workers would otherwise have to go to, receive treatment, and return to work from any of the neighbouring hospitals or surgeries. In the same way the time of general practitioners is also saved by carrying out their routine treatments at the Centre instead of at individual surgeries. These practices have, as well as their obvious functions, the important one of making the Centre a close ally of the local general practitioners and consultants. It takes its place in the pattern of preventive medicine and of potential research not only in a selected group but of the whole community in its relation to several environments.

All photographs by Shell Photographic Unit

British Occupational Hygiene Society

The Use and Abuse of Protective Equipment

C. N. DAVIES

This conference was held in London on November 4, 1955. It was divided into four sections dealing respectively with the protection of the lungs, skin, eyes, and feet.

The morning session was opened by Mr. S. H. Wilkes, Senior Chemical Inspector of Factories, who gave a warning that the canister respirator, which was an excellent device in its own proper field, should not be used inside closed spaces where there might be high concentrations of poisonous substances or a shortage of air. An air line respirator fed with fresh air from a hand pump should always be used in such situations.

Papers on the protection of the lungs were presented by Mr. A. C. Peacock, of the Chemical Defence Experimental Establishment, Porton, and by Mr. J. Whittaker, of the Central Safety Department, Imperial Chemical Industries, Ltd.

Mr. Peacock stated that protection of a worker by means of a respirator must be regarded as a second-best substitute for tackling the hazard at its source.

The design of a respirator had to be considered as a whole ; vision, comfort, and speech had to be ensured and must be combined with durability and low breathing resistance. It was also important that the hazard against which it was to be used should be fully specified so that the most appropriate kinds of filter could be incorporated.

Gases and vapours were absorbed by activated charcoal which was usually suitable unless the molecular weight was too low. It was then necessary to impregnate with catalysts. Ammonia, oxides of carbon, and the oxides of nitrogen needed special absorbents. Aerosols were taken out by fibrous filters in the form of paper, carded lap, or felted fabric.

The fit of the facepiece was important as contact with the skin without the use of excessive pressure had to be maintained when the head was moved violently. It was easier to fit well full-size face masks going under the chin than small ones covering only the mouth and nose. It had been established that a plain polished surface gave better contact with dry skin than a roughened one.

The larger the protective appliance, however, the greater was the aversion of the worker to wearing it and the greater the risk of its being discarded. Workers in the older industries were difficult to discipline in this matter and propaganda based on fear or a humorous appeal was deprecated. The atomic energy industry was exceptional and fortunate in that its employees were well aware of the danger of their working conditions and cooperated fully in the precautions advised.

Mr. Whittaker emphasized that the prevention of lung injuries began with good design of buildings, processes, and plant, followed by the use of sound operational methods and the training and control of personnel. Personal protective equipment was a last resort, required by law in certain operations.

The discussion on protection of the lungs was opened by the President of the Society, Dr. J. M. Rogan, Chief Medical Officer of the National Coal Board. He mentioned one difficulty when canisters were used in protective masks. This was the lack of any visible indicator of the amount of useful life left in them, which depended not only upon the concentrations of gas in which they had been used but also upon the rate of breathing.

In assessing the danger of a dust it had to be remembered that the intratracheal toxicity was usually many times greater than the oral figure.

The cooperative attitude to protective measures of workers in atomic energy establishments was put down to their being accustomed from the start to hazards outside human perception, to their being given full information about the risks, and to good discipline.

The second section of the conference, on the protection of the skin, consisted of papers by Professor J. R. Squire of the Department of Experimental Pathology, University of Birmingham, and Dr. A. Lloyd Potter, of the General Chemicals Division of Imperial Chemical Industries Ltd.

Professor Squire gave an account of the structure of human skin and explained the mechanisms of various kinds of damage. The outer layer of the epidermis was chemically inert and resistant to acids but it swelled in alkalis and, being a protein, could react with, absorb, and transmit substances which contained $-NH_2$ or -SH groups. It was also liable to be destroyed by reducing agents and cracked if dried excessively. Normally loss of water was impeded by the fatty surface film so that removal of this by solvents might result in "chapping". The fatty layer was replenished in about two hours.

Sheer mechanical abrasion was encountered in some occupations. The horny surface of the skin was constantly replaced from the germinal layer, and if the rate of abrasion was too high and gloves could not be worn, a shorter shift was the only remedy.

Chemical agents acting as irritants produced their effect in 48 hours or less, but sensitizing chemicals might produce intense reaction on a second exposure 10 or 14 days after the initial one. Once sensitized to a specific chemical a man could react to minute traces of it. Complete removal to another occupation was necessary in these cases. Carcinogens required years to produce even the pre-cancerous effects and the peak incidence of the cancers was 20 to 30 years after exposure; their action was dependent on repeated exposure and could be reduced by lessening the amount of carcinogen reaching the skin.

Mechanical handling avoided many risks but was not applied to many jobs of low economic value; for such occupations personal protection was necessary. The organization of protective equipment should include provision for inspection, repair, and cleaning. There was a risk of transferring contamination to the person when soiled clothing was removed. Barrier creams could be relied on to exclude a toxic chemical only in exceptional instances.

Dr. Lloyd Potter stated that although occupational dermatitis was not notifiable to the Chief Inspector of Factories over 3,000 cases were voluntarily reported each year. In order to affect the skin a substance must wet it, either by being itself in liquid form or by solution in perspiration or other liquid on the surface of the skin. Water, alcohol, and weak acids did not greatly affect the cells of the skin, but they were easily damaged by alkalis and fat solvents which penetrated the natural greasy barrier and reached the sebaceous layer. Dermatitic agents fell into two groups: primary irritants and skin sensitizers. It was almost impossible for the employee who was liable to contract dermatitis to be detected before employment by medical examination, but regular examination subsequently was very important.

The ideal method of protection was by completely enclosing a process, but this could be nullified if, for example, the final product was packed by hand. Protective clothing should be neat and effective; it should not add to the risk of contracting dermatitis by causing sweating and chafing. Inspection, laundering, and repair should be organized and the wearer should be educated by precept and example. Proper washing facilities were important and barrier creams had their place if only because they encouraged washing.

In the discussion Professor Squire stated that the *p*H of the skin was about 4·5 or 4·6, partly due to carbon dioxide transpiration and partly to free fatty acid in the surface film. The fatty acid layer gave important protection and might be disturbed by long exposure to alkalis.

Introducing the speakers in the afternoon session of the Conference, Sir Harold Roberts, Chief Inspector of Mines, stated that mining was the most dangerous of all major industries. The death rate in coal-mining was nine times as high as the death rate in factories. The absolute figures were improving but relative to employment in factories there seemed to be little change. Major catastrophes were responsible for only a very small proportion of the casualties in mines; the major toll was taken by falls of roof and transport operations.

Injuries to the feet and ankles were common and the wearing of safety boots bearing the British Standard mark was encouraged, but there was a long way to go before they were universally adopted by all workers. It was even more difficult to get the underground worker to wear goggles. Even the slightest interference with vision was trying and could create additional risks. Improvement in lighting had, however, made a real contribution to safety and to the prevention of miner's nystagmus.

The President stated that the fatal accident rate in British mines was half the German rate, one-third of the American, and lower than that of any other mining country except the Netherlands.

Dr. D. R. Campbell, Director of Research, Birmingham and Midland Eye Hospital, read a paper on "Ocular Injuries and Hazards". She stated that eye injuries constituted 4·26% of all industrial accidents; most of them were due to flying particles and were often preventable. In addition, there were thousands of minor injuries from the same cause which were not reported because they caused less than three days' absence from

work. The risk was greatest in metal foundries and heavy engineering.

In the matter of protection, it was disheartening to find so many goggles which steamed up, restricted vision, and were uncomfortable to wear. The issue of goggles to every individual worker, each being given a choice of four designs, in a large engineering factory had demonstrated that a great decrease in accidents could be achieved.

Ocular injuries could often be repaired now that the complication of infection was largely absent; all the same, even a small scar in the centre of the cornea could cause disability by dazzle in artificial light. Eye protection should be compulsory in occupations involving striking and chipping with a metal tool. Burns from molten metal and chemicals, especially caustic soda, were dangerous. Immediate irrigation with water might be the means of saving the eye.

In his paper on "Eye Protection, Types of Equipment and their Use", Mr. G. V. Thom, Plastics Division Safety Officer, Imperial Chemical Industries Ltd., explained the legal obligations to provide protection for the eyes against certain hazards which are laid down by the Factories Acts and are required by Common Law.

Built-in protection upon the machine, rather than upon its operator, was desirable, and he showed illustrations of typical methods of applying this principle.

Personal eye protection included spectacles, which could have prescription lenses made in safety glass, eye shields covering a part or all of the face, and goggles. Protection against infra-red rays was needed in welding and against ultra-violet as well in electric arc welding, which necessitated screening the whole of the face. British Standards for protective appliances were in existence. Persons with monocular vision should be given special attention because of the risk of total blindness, particularly because the existence of their disability might be unsuspected at the works.

It was suggested in the discussion that old hammers and chisels with mushroomed heads were a common source of intraocular foreign bodies; the risk was decreased by regular replacement of worn tools. Limitation of the field of vision by goggles, especially downwards, and interference with binocular vision discouraged their use. Other points raised were the need for safety glass, plastics which were not inflammable, and the fact that coloured glass which gave protection against radiation from flame welding might be useless for arc welding.

Mr. H. Bradley, Director of the British Boot and Shoe and Allied Trades Research Association, spoke first on the protection of the feet and was followed by Mr. H. E. Thomas, Head of the Safety Department of the Ford Motor Company, Dagenham. Mr. Bradley outlined the steps which had led to the drafting of a British Standard for safety footwear. These have a steel toe-cap which has to resist an impact test. In the experience of Mr. Thomas the standard safety boot or shoe did, in some instances, give protection, but there were still many cases of toe fracture while wearing them. He suggested that the metal toe box be extended further back, even though this meant it would be unable to pass the impact test.

ORDINARY SCIENTIFIC MEETING

An ordinary scientific meeting of the British Occupational Hygiene Society was held at the London School of Hygiene and Tropical Medicine on June 20. Dr. T. Bedford was in the chair and some 50 members were present. A brief summary of the papers contributed to this meeting is given below.

Exposure to Trichloroethylene during an Industrial Degreasing Operation

D. E. Hickish, J. H. Smith, and Joan Bedford reported the results of a routine examination of a typical degreasing operation using a toxic solvent. The methods of collection and subsequent analysis of air samples were discussed. Urine specimens and blood samples were examined and clinical examinations performed. Concentrations of the solvent in air in excess of those recommended as the maximum allowable were discovered. Some abnormalities in the blood and urine specimens and mild clinical symptoms were noted. There were, however, no major pathological disorders.

The need for routine supervision of potentially dangerous operations was emphasized.

Procedures relating to the Safety and Hygiene of Persons Handling Tetraethyl Lead in the U.K. and Overseas

P. R. Boyd outlined the tetraethyl lead hazard with reference to the areas and spheres of activity in which this compound is handled. The general administration and supervision of those exposed to the potential absorption of this material were discussed in relation both to the manufacturers and to the customers and users.

The detailed control in terms of plant hygiene and human monitoring was briefly described, showing the combined use of clinical supervision and of analysis of human excreta and of the atmospheric environment.

The tetraethyl lead industry is a good example of one involving severe occupational hazards which can be controlled by detailed supervision.

A Small Outbreak of Mercurialism in Laboratory Workers

J. F. Erskine described a small outbreak of mercury poisoning in laboratory workers analysing mine air samples. Symptoms were minimal but the probable cause was confirmed by the finding of mercury in the urine. The risk was partly eliminated by cleaning the laboratory and improving the working conditions. It was finally eliminated by a change in the method of analysis so that open vessels of mercury were no longer required.

The outbreak demonstrated the risk of chemical poisoning to laboratory workers when, by increasing demand, ordinary laboratory techniques are converted to mass production methods, and the fact that the risk can be eliminated by the normally accepted methods of plant hygiene.

Assessment of Silicotic Fibrosis by a Chemical Method

B. D. Stacy said that the development of fibrosis in tissues could be studied qualitatively by histological procedures. For assessing the extent of fibrosis throughout an entire organ, *e.g.*, lung or liver, these techniques are somewhat limited ; a more quantitative approach to the problem has been made by the application of chemical methods.

" Fibrosis " implies the formation of the fibrous proteins reticulin and collagen. A chemical method has been adapted for determining them, and a relation has been sought between total tissue collagen and the severity of the fibrosis. The chemical determination is based on the estimation of hydroxyproline, collagen being unique in containing a high proportion (14%) of this amino-acid. By the use of standard chromatographic procedures the protein under investigation was also checked and studied in relation to its general amino-acid composition, to ensure that it was similar to collagens previously described in the literature.

The method has been applied to three series of animal experiments designed to study (1) the fibrogenic effect of different forms of free silica ; (2) the fibrogenic effect of varying amounts of tridymite ; (3) the effect of cortisone on developing and established silicosis. In these experiments the collagen values, determined chemically, have strongly supported and quantitatively extended histological observations on the development of fibrosis.

A Survey of Respiratory Symptoms and Pulmonary Disability in a Random Sample of the Population of an Industrial Town

I. T. T. Higgins said that respiratory disability in coal-miners was usually due either to pneumoconiosis or to bronchitis. While the former is caused by exposure to dust at work, the latter is common in men who have never worked in dusty occupations. Before incriminating the occupation it must be shown that the prevalence of the disease is higher in miners than in the non-mining population from which they are recruited.

In November, 1954, the Pneumoconiosis Research Unit carried out a survey on a random sample of the male population aged 55 to 64 of Leigh, Lancashire. The main purpose was to compare the prevalence of respiratory symptoms and pulmonary disability found in miners with that found in non-mining controls.

The respiratory symptoms were recorded, using a questionnaire, and occupational histories were taken to enable the required grouping to be effected. The ventilatory capacity was assessed by a single breath method.

Chest radiographs revealed those men with pneumoconiosis and other significant pulmonary disease. It was found that miners and ex-miners had significantly more chronic bronchitis and a significantly lower ventilatory capacity than men who had never mined.

The prevalence of bronchitis was lower in miners whose radiographs showed pneumoconiosis than in those with normal radiographs. In contrast the ventilatory capacity was higher in the men with pneumoconiosis than in those without.

Some Factors Affecting Absence Rates

R. B. Buzzard said that absence rates were variously used as indices of health, morale, or efficiency in working groups, or as criteria for testing selection procedures, training methods, or work processes ; but controlling the many factors which affect them in order to examine one was extremely complex.

Assumptions about the meaning of absence rates need questioning. " Lost " time depends on the time people are asked or permitted to work ; increased attendance may accompany increased average lost time. Early researches of the Industrial Health Research Board showed the amount and nature of work to be among the causes of differences in attendance ; little research has been done on the optimum attendance for different tasks. Apart from effects of direct fatigue, a man who is off colour may be fit to attend for light work but not for heavy physical tasks. Conventional absence rates may be markedly affected by the behaviour of a few people ; they do not necessarily reflect the behaviour of the group. Short absences contribute less to average rates than long ones but most people agree that they upset production more ; lost production cannot be calculated from average lost time rates.

The study of absences is medically important but handicapped by ignorance of the attendance which should be expected for particular jobs in particular conditions. Systematic observation of men at work is needed as a complement to any study of lost time statistics.

CARL M. PETERSON, M.D., 1899–1955

"Pete", as we affectionately knew Carl Peterson, believed in organized medicine. He believed in that section of our code of ethics which says, "The avowed objective of the profession of medicine is the common good of mankind." He knew that it applied equally to workmen and bosses, and he set his goals in this direction. Little by little he made progress, some years more, some less. He knew that the sociological progress of medicine is slow and depends largely on the worthiness of objectives clearly understood and constantly in sight. During the bad years when little progress was made, we never heard him complain. Nor did he blame anyone, unless himself. He quietly found another angle of approach and pointed it out to us. During the good years when much progress was made, he was so surprised and delighted that he had a ready list of those deserving credit, including everyone but himself.

Carl Melancton Peterson was born in Rockford, Ill., on December 6, 1899. He qualified at Augustana College in 1921, and proceeded M.D., University of Minnesota, in 1927. After hospital appointments and some years in general practice he joined the A.M.A. staff as hospital inspector in 1930 and became Secretary of the Council on Industrial Health in 1938. He was a member of a great many committees and public bodies, and of the Editorial Board of the *Archives of Industrial Health*. He served as Chairman of the Committee on Occupational Health Services of the World Medical Association and was Secretary of the Interim American Board of Occupational Medicine, which led to the inclusion of occupational medicine in the American Board of Preventive Medicine. He was the author of numerous articles on educational aspects of industrial health.

A few of us will long remember our last evening with Carl. The Council meeting [of the A.M.A.] was over, and he could relax before flying next morning to other Council business. He never admitted that he had done a good job. He still thought and talked only of the job ahead. But we who knew him saw that he was at peace with himself.

Abridged from a full obituary in *Archives of Industrial Health* by kind permission of the author, Dr. W. P. Shepard, and publishers.

BOOK REVIEWS

Digest of Statistics Analysing Certificates of Incapacity, 1951 and 1952. London: Ministry of Pensions and National Insurance. 1954.

This is the second report from the Ministry of Pensions and National Insurance analysing information on medical certificates for sickness or injury benefit. Its basis is mainly a sample of those certificates relating to 1951, but some information is also given for 1952 sickness, new information for 1952 injuries, and a Table showing the effect of the influenza epidemic of the winter of 1952–53 on the number of claims for benefit. For readers of this journal the new Tables analysing sickness by occupation will be of special interest.

"The statistics do not purport to give any measure of the morbidity of the whole population or of a representative sample of it." Briefly, for sickness benefit, they cover the working population between the ages of 15 and 68 for men (15–63 for women), excluding those drawing retirement pensions, members of the Armed Forces, non-industrial civil servants with illnesses of less than six months' duration, about half the married women, and some 20,000 self-employed persons with small incomes. Chronic sickness starting in the pre-1948 period may be somewhat under-represented. For injury benefit, covering accident or prescribed disease, the self-employed are excluded but married women are included.

In 1951 there were 7½ million new claims for sickness benefit; just over 7 million "spells" of sickness ended during the year, and there were 284,700,000 days of certified incapacity. The figures of days lost are based on working days, so that during the year 910,000 man-years were lost from sickness alone out of a force of 20 million at risk, that is, approximately 4½% of the labour force per year. (In the same year about 540,000 man-years were lost through industrial stoppages so that sickness accounted for almost 80% more lost time than stoppages.)

For men, 24% of the periods ending in 1951 were due to influenza, 10% to bronchitis, and 8·6% to arthritis and rheumatism. When days lost are considered, the same three groups come at the top of the list, with bronchitis accounting for 11% of the total days' duration, influenza 10%, and rheumatism and arthritis 7½%. Respiratory tuberculosis follows with 6%. For women the same four groups of disease head the list. Of the total days lost, rheumatism and arthritis account for 9%, influenza for 8%, and bronchitis and respiratory tuberculosis both for 7%.

In addition to the time lost due to sickness, there were 766,900 new claims for injury benefit, of which 697,500 were due to industrial accidents and 43,600 to prescribed disease. When considering days lost due to injury claims, it must be remembered that injury benefit cannot in any case be paid for more than six months from the date of the accident or the development of

the disease. If incapacity lasts longer than six months, sickness benefit is payable and so will appear in the Tables as one six-month spell of injury benefit and one spell of sickness benefit for the remainder of the period. Moreover, the report points out that " because in the early stages they are not incapacitating diseases, pneumoconiosis and byssinosis do not attract injury benefit though if they progress to an incapacity stage they do attract sickness benefit ".

Tables 7 to 10, showing periods of sickness by occupations, are worth detailed study by all industrial medical officers. (The relevant populations at risk for men are in Table 91.) The total male sickness rates follow the pattern expected, persons in mining and quarrying having almost two and a half times the rate for all men, followed by unskilled workers and foundry workers both with 1·4 times. At the " healthy " end of the scale there are also foremen and overlookers in engineering and allied trades, fishermen, agricultural workers, persons in entertainment and sport, and electricians, " administrators, directors and managers ", and several other such groups for whom these figures no doubt do not reflect their true sickness rate. The figures are not standardized for age and this no doubt accounts for some of the differences shown. The Tables also give figures for the most frequent causes of sickness within the different occupations.

Industrial accidents are classified by industry and external cause. About a quarter of accidents to men are caused by a " blow from a falling object " and another fifth from accidental falls. For women falls account for most absences, followed by accidents caused by " cutting and piercing instruments ".

Many other points of interest arise from other of the 92 Tables which include analysis by age, geographical region, and duration of sickness.

The report has a full index to the Tables and a good explanatory introduction with full definitions of the sources, scope, and meaning of the statistics, and contain warnings about their possible misinterpretation or misuse. It seems a pity that the report has not been published generally because of the possibility of unintelligent use by people unwilling to read or digest the seven pages of the introduction. One wonders if the compilers of the first reports on mortality at the Registrar General's office met with the same difficulties of unintelligent use or intelligent misuse over a century ago. Fortunately it did not stop the wide circulation of their reports. It is to be hoped that future reports on morbidity by the Ministry of Pensions and National Insurance will take the form of an H.M.S.O. publication available for all.

In the meanwhile, enquiries for copies or information should be addressed to the Medical Department or the Statistics Division of the Ministry of Pensions and National Insurance at 10 John Adam Street, London, W.C.2.

N. M. GOODMAN

Report on the Health of the Army, 1951 and 1952, 24/General 3756. The War Office. London : H.M. Stationery Office. 1954.

The present report covers two years and is of great interest. The Pulheems system, which underwent a severe test during the Korean War and the recall of reservists (Z men) to training, has again proved its value. Employment standards can be altered without altering the basic assessment, to conform to any necessity, and in these two years they were markedly altered on account of the major shortage of man-power. For the first time Grade III men were conscripted and formed almost 4% of the man-power available. Not unnaturally the number of discharges on medical grounds subsequently increased.

A further interesting innovation was made in 1950 when sergeants were posted to civilian medical boards to undertake simple intelligence testing and to report their findings to the chairmen. As a result discharges on both medical and psychiatric grounds fell appreciably although the report does not state what proportion was lost initially. If there is no serious discrepancy between original rejections and subsequent discharges the method is of obvious value.

In males about a third of discharges on medical grounds were due to psychiatric conditions headed by anxiety neurosis, hysteria, and psychopathic personality disorder. In females about half the discharges were on psychiatric grounds. In both sexes pulmonary tuberculosis was the next highest cause of discharge, followed, in males, by peptic ulcer and accidents.

Research continued in a number of diverse subjects from clothing to insecticides and on all matters of environmental hygiene. The statistical section again takes up a large proportion of the report and is a clear summary on the various aspects of health. With the improvements in general hygiene the Rickettsias are perhaps the greatest potential source of danger because of the assumption of epidemic form in large-scale troop operations.

J. P. W. HUGHES

Studies on Expenditure of Energy and Consumption of Food by Miners and Clerks, Fife, Scotland, 1952. By R. C. Garry, R. Passmore, Grace M. Warnock, and J. V. G. A. Durnin. (Pp. 70. 5s.) Medical Research Council Special Report Series No. 289. London : H.M. Stationery Office. 1955.

This reports a piece of classical physiological research. The fact that such studies are rare nowadays makes this one all the more valuable, but its rarity is, perhaps, understandable when we read that in order to do it at all more than 30 investigators were occupied for a period of five weeks. During this time they were able to study 19 coal-miners and 10 colliery clerks.

The work that was done was to estimate the energy expenditure of the men by measuring their oxygen consumption with a Kofranyi-Michaelis respirometer while they were doing different kinds of work during their shift in the mine and also during all their home occupations, standing, sitting, lying, washing, dressing, cycling, and playing golf—these were Scottish miners. During working hours an observer accompanied each miner or clerk the whole time and recorded what he did in a book ruled into separate columns for every minute of the day. When the men were off duty they themselves

filled in a similar book and the observers had to be content with paying a daily visit to each one's home. In parallel with the collection of information about energy expenditure, food intake was studied by weighing everything eaten throughout the week of survey. The calorie value of the diet and its nutritional composition were calculated from food Tables.

The thoroughness of the whole investigation makes it a model of its kind. Information reported in detail of the energy expenditure for different kinds of activity represents a valuable addition to the literature, where, all too often, figures derived from long-forgotten experiments have been copied from one authority to another. An interesting observation arising from a statistical review of the data is that there is a highly significant relationship between the expenditure of energy for many kinds of activity and the body weight of the men carrying them out. This holds not only for walking, where a man is obviously carrying himself about, but also for such work as hewing coal and " girdering ".

The average estimated energy expenditure of the miners of 3,660 calories a day compares with 3,820 and 3,360 calories for German miners studied in 1943–44 and with 4,070 calories estimated by Moss for English miners in 1935. It is interesting to notice that in comparing the nutritional value of the diet with the British Medical Association's estimate of what a good diet ought to provide, no strictures were passed on the vitamin C content, which used to be a traditional target for doubts. The only possible weakness suspected in some few instances was as to whether there was enough riboflavin.

More and more in up-to-date industries machines are used in place of men to do mechanical work. In this coal-mine in Fife the energy expenditure of clerks and miners outside their working hours was almost identical. At work, however, the miners used 1,750 calories a day and the clerks 890 calories. Yet, as the authors point out, for three hours out of the eight-hour shift the miners could do no work because for much of this time breakdown of the mechanical haulage system prevented their doing so. Moss assessed how low was the mechanical efficiency of a man shovelling coal while crouching in a mine. No such calculations were made in the present study. Nevertheless, in addition to the debt we owe to the authors of this valuable piece of physiological investigation, we shall owe them more if it enables us to relate in terms of modern engineering logic the human energetics of coal-mining to the job to be done.

MAGNUS PYKE

The Protection of Workers against Ionising Radiations. Report submitted to the International Conference on the Peaceful Uses of Atomic Energy, August, 1955. (Pp. 66; 11 figures. 50 cents; 3*s*.) Geneva: International Labour Office. 1955.

It is greatly to the credit of the International Labour Organization that it was able, at short notice, to produce this useful summary of the problems of radiation protection in time for the International Conference on the Peaceful Uses of Atomic Energy in August, 1955. Although little originality can be claimed for the material of the report, it is a welcome addition to the general literature on this subject and it will be of immediate interest, particularly to those who are contemplating these problems for the first time.

From an outline of the hazards of radiation work, the report goes on to consider the essential problems of radiation protection and it describes the special precautions appropriate to a range of the more common applications of radiation in industry, medicine, and agriculture. With the rapid expansion of the uses of radioactive substances there is growing concern about the safe transport of these materials, and the report contains an interesting section on this subject.

The authors are to be congratulated upon the fact that they have refrained from adding to the confusion which surrounds the genetic implications of irradiation. The list of references is surprisingly brief and will barely satisfy the keen student of this subject. It is apparent that the International Labour Organization and International Commission on Radiological Protection have many common interests; even now, they are being joined by the World Health Organization. It may be that the time is not far distant when some formal agreement will be required to clarify the relative responsibilities of these three bodies.

A. MACLEAN

Clinical Orthopaedics, Number Five. Edited by Anthony F. DePalma. (Pp. viii + 242; 158 figures. 40*s*. per copy subscriptions; single copies 60*s*.) Philadelphia and Montreal: J. B. Lippincott Company; London: Pitman Medical Publishing Co., Ltd. 1955.

In 1953 a group of distinguished American orthopaedic surgeons launched an unusual periodical, of which this is the fifth issue. Each number appears in book-form, cloth-bound, and so far two have appeared each year. Each has been divided into two sections, a collection of essays on one particular orthopaedic topic being followed by a series of papers on miscellaneous orthopaedic subjects.

This fifth issue, which was published in late July, 1955, devotes its first section and three-quarters of its pages to 17 papers on backache and associated problems. There can hardly be a subject in the whole field of medicine which gives more difficulty more often to more practitioners in almost all fields, and it must be said at once that these essays will prove stimulating to anyone who is interested in the back. The orthopaedic surgeon, to whom they are primarily directed, will find much help in these pages and this issue, like its predecessors, is an essential book for any reasonably comprehensive orthopaedic library.

Your reviewer feels bound to doubt the real practical value, though not the interest, of this publication to the industrial medical officer. Four of the papers in the first section are mainly or wholly concerned with operative techniques, four others offer rather personal views on physical treatment and on manipulation, another is devoted to the evolution of the back, and one more is purely biographical. There is sound sense and good advice in T. A. Willis's paper on " The Inadequate

Back " (which, as he so rightly says, commonly occurs in the inadequate individual) but the essay is superficial and rather sketchy. It should have been twice as long as it is to let the author develop his opinions for our benefit. The same sketchiness is strikingly illustrated by a contribution with the inviting title of " The Industrial Back—A Clinical Study ", in which a general review of the problem and accounts of the mechanism of back injury, of " anatomic diagnosis ", " roentgenograms ", examination and treatment occupy only three pages.

The second section deals with advanced orthopaedic problems in a stimulating and interesting manner. Its contents are, however, well outside the field of industry.

It is only fair to say that though this issue has much for the orthopaedic surgeon and little for the industrial medical officer, the proposed twelfth issue in this series, which promises to deal with rehabilitation, may well be of interest and importance in industry.

D. Ll. Griffiths

Dictionary of Organic Compounds. 4 vols. Edited by Prof. Sir Ian Heilbron and H. M. Bunbury. (£28 the set; £7 10*s*. 0*d*. per volume.) London: Eyre and Spottiswoode. 1955.

This dictionary was first published in three volumes between October, 1934, and December, 1937, and has an established place in the libraries of chemistry departments. The new edition has been completely revised and brought up to date to the end of 1950 (and results quoted till early 1953) by a number of distinguished organic chemists, and is indeed a monumental work comprising 3,031 pages in all. Over 2,500 compounds appear for the first time as principal entries.

The constitution and physical and chemical properties of the principal carbon compounds and their derivatives, together with the relevant references to the literature, are concisely presented in alphabetical order, and all the data are readily accessible with abundant cross references. The Editors have set out to provide information on the many new organic compounds, both natural and synthetic, which have come into prominence in recent years as the result of the rapid expansion in all branches of chemistry and in the field of biochemistry. Data on each compound include its sources, physical properties, chemical properties (typical reactions, analytical tests) with derivatives described under separate sub-headings.

In regard to its use by workers in the field of industrial medicine and toxicology, two minor aspects of presentation may appear confusing. The continual employment of the letters d and l to designate optically active compounds, rather than the modern connotations D, L, +, −, could cause confusion in the minds of readers not familiar with isomers of sugars and amino-acids. Also, where a number of isomers obtain for one molecular formula, the first member only is described. This leaves gaps in information on compounds of biochemical interest, *e.g.*, porphyrins, haems, and bile pigments, for which knowledge of isomer type has important implications in the industrial medical field.

These minor criticisms apart, it is obvious that this dictionary represents a successful up-to-date attempt to cover in reasonable compass the whole field of carbon compounds. As such it will be invaluable to libraries of chemistry departments and research institutions to provide a repository of fundamental knowledge available not only to chemists but to specialists in such other fields as industrial medicine.

J. E. Kench

Textbook of Rheumatic Diseases, 2nd ed. Edited by W. S. C. Copeman. (Pp. viii + 754; 464 figures. 52*s*. 6*d*.) Edinburgh and London: E. & S. Livingstone. 1955.

Since its introduction in 1948 this book has come to be accepted as the standard work on rheumatic disease in this country and has earned world-wide recognition. Though it is only seven years since the appearance of the first edition, the introduction of steroid therapy just after the initial publication has necessitated much rewriting and some rearrangement of the earlier chapters. A new chapter on the adrenal hormones has been added, and this gives a short but useful review of this side of the problem. Much information on the use of cortisone and A.C.T.H. has also been included in the chapters on rheumatic fever, rheumatoid arthritis, gout, and other rheumatic diseases. Two chapters on pain have been merged and form a most useful introduction to the problems of skeletal symptomatology as well as a valuable source of reference to the segmental distribution of deep pain. In the chapter on rheumatic fever both the old salicylate and the newer hormone methods of treatment are critically considered, and indeed throughout the book a cautious attitude is adopted towards the new methods of treatment and affords a valuable corrective to current enthusiasm. In the treatment of rheumatoid arthritis the importance of general and local rest to joints combined with maintenance of muscular power and optimal joint posture is rightly stressed. It may be questioned whether the advice to withhold chrysotherapy in the first few months of treatment is altogether in the best interests of the patient as it is in this stage that adequate suppression of the process would appear most likely to be beneficial. No reason is given for recommending the relatively toxic aqueous solution of sodium aurothiomalate, and the question of high or low dosage of gold is not discussed, only low doses being recommended. The value of other methods of treatment is critically reviewed and a well balanced account of the treatment of rheumatoid arthritis is provided.

There is a useful chapter on the rarer arthritic syndromes which will delight the collector of eponyms, and there are excellent short chapters on radiology and radiotherapy. The chapters on physiotherapy give a useful account of the methods in current use and at the same time avoid the excessive claims sometimes made for this type of treatment.

Rheumatism in industry is considered very briefly. Following a discussion of the toll of rheumatism as a whole in the employed population, reference is made to the high prevalence rate in certain specific occupations —steel, mining, and transport. The importance of cooperation between industrial medical officers, employers,

and production managers to ensure that mechanization reduces and does not increase the rheumatic hazards is stressed, though it is admitted that much more research into the influence of occupation is required if effective steps are to be taken. Unfortunately, intervertebral disc degeneration, which forms such an important part of the rheumatic hazard, particularly in heavy industry, is not very fully discussed in any part of the book. Its importance has, however, been recognized only comparatively recently and it will, no doubt, receive greater prominence in future editions.

As inevitably happens in a book by many contributors of varying disciplines, there is not only much repetition but also some disagreement, particularly on the treatment which may be pursued. This, however, is not altogether a disadvantage in the present state of our knowledge.

The chapter on the prevalence of the rheumatic diseases is valuable but tends rather to neglect recent work.

As in the first edition the illustrations and radiographs are beyond reproach, and this book can be confidently recommended to the industrial medical officer who requires an authoritative guide to present-day knowledge of the rheumatic diseases.

J. S. LAWRENCE

Vitamins in Theory and Practice, 4th ed. By L. J. Harris. (Pp. xxii + 366 ; 103 figures, 107 tables. 35*s*.) London : Cambridge University Press. 1955.

The first edition of Dr. Leslie Harris's book appeared in 1935 and contained a survey of the progress made in the study of vitamins to that date. It rapidly became accepted as a standard book on the subject, reaching the third edition before the second world war.

The period during and since the war saw great advances in this subject, many further substances and groups of substances being added to the list of vitamins, while knowledge of the accepted vitamins was greatly extended. The fourth edition has, in consequence, been enlarged and extensively rewritten to accommodate recent advances. Perhaps the most startling change is the rise (from six to 20) in the number of vitamins discussed.

Dr. Harris has three separate but closely intertwined stories to relate : first, the initial experimental work involved in the recognition of certain diseases as being due to a dietary deficiency and of the discovery of the foods necessary to restore health ; secondly, the large scale introduction of these foods or of vitamin-rich extracts to effect the eradication of the diseases in the areas affected ; finally, the isolation, identification, and synthesis of each vitamin by the organic chemist, allowing some insight to be obtained into the mode of action of the vitamin in the body.

The author has treated this complex subject in a masterly fashion, including an immense amount of factual material in a lively narrative. *Vitamins in Theory and Practice* is eminently suitable for the non-specialist without being superficial. The industrial medical officer dealing with dietary problems in industry and with the special treatment of patients subjected to particular hazards, *e.g.*, chlorinated hydrocarbons, T.N.T., etc., should find it particularly useful.

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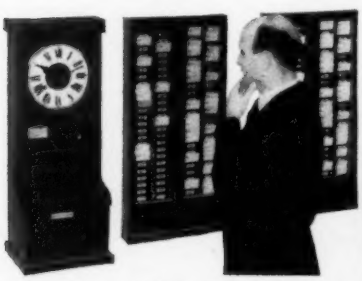

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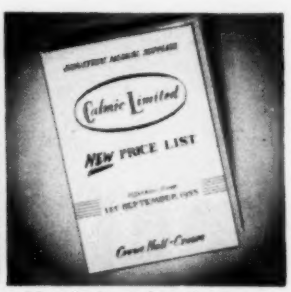

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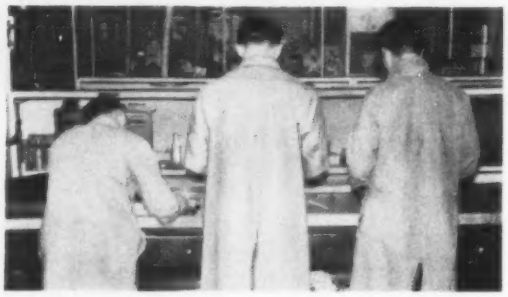

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